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COMING events among the state associations forecast a season of educational opportunities and furnish the only means of solving the problems affecting the security of the veterinary profession.

PARTICIPATING in these events is an obligation leading to one's pleasure and profit and to a sounder veterinary service for the American people.

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Vol. LXXXIII, N. S. Vol. 37 DECEMBER, 1933 No. 6

HOUSE OF REPRESENTATIVES

During the next few months, associations in about half the states will hold meetings. It will be in order for those that have not already done so, to provide for representation at the first meeting of the A. V. M. A. House of Representatives in New York, next August. As has been announced previously, this body will take over the functions formerly performed by the membership at large at the general business sessions of each annual convention, with certain exceptions, such as the election of officers.

Thirty-five state associations have approved the plan of affiliation with the A. V. M. A., upon which the change was contingent. The associations in the following states have not yet voted to affiliate: Alabama, Arizona, Arkansas, Indiana, Iowa, Mississippi, New Hampshire, New Mexico, Rhode Island, South Dakota, Vermont and Wyoming.

It is still possible for these state associations to affiliate and have representatives at the 1934 meeting in New York.

Inquiries have been received as to just what effect affiliation would have on the state associations. These questions have suggested that there is some fear that a state association would lose its autonomy. This is not the case. Each state body will continue to function as in the past, although it is hoped that the House of Representatives will bring about a greater degree of cohesion

among the state associations than there has been in the past. There have been entirely too few instances of associations in neighboring states getting together and pooling their efforts in a common cause.

Why do we not have more joint meetings of two state associations? Such gatherings could be made to be highly attractive and profitable. Each summer for a number of years, the associations in Oregon, Washington and British Columbia have held a joint meeting. Last summer the North Carolina and Virginia associations met together and had a very successful meeting. The New England Veterinary Medical Association, on a slightly different basis, is really a union of the six state associations in New England.

Quite frequently of late years some remark has been heard to the effect that we have too many veterinary associations nowadays. Such a comment would be more accurate if it were to the effect that there are too many veterinary meetings. This situation could be remedied very easily by developing the plan for joint meetings. For example, why would it not be feasible for Minnesota and Wisconsin to get together on a plan for holding their summer meetings together, alternately in the two states, and preferably in cities along the borderline? Or Missouri and Illinois? Or Texas and Oklahoma? These are just suggestions to illustrate the idea.

INTERNATIONAL VETERINARY CONGRESS

Plans for the Twelfth International Veterinary Congress are taking form rapidly. The big task of selecting the reporters was completed about two months ago and the next step was to forward the official notifications to the more than one hundred prominent veterinarians in all parts of the world. If you could have dropped into the A. V. M. A. office early in October, you would have seen an unusual activity there—letters being written in the four official languages of the Congress—English, French, German and Spanish. This has become a daily occurrence since the arrival of Mr. Ernst Haux, about the middle of November. Those who attended the Eleventh Congress in London, in 1930, will remember Mr. Haux as a member of the staff assisting Dr. Fred Bullock, general secretary of the Eleventh Congress. Mr. Haux will be located in the A. V. M. A. office until some time after the Congress.

Of especial importance at this time is the announcement that President Roosevelt has accepted the invitation of the Organizing

Committee to act as Patron of the Twelfth International Veterinary Congress. It is hoped that next month we will be able to announce that another high government official will act as Vice-Patron.

In another part of this issue of the JOURNAL will be found the personnel of the Organizing Committee as augmented up to the time of going to press. Subcommittees to handle the numerous details will be appointed and announced from month to month. Beginning with the January issue, the JOURNAL will have a separate department each month, devoted to news concerning the Congress. Watch for it.

12th International Veterinary Congress
New York—August 13-18, 1934

CONCERNING DUES

Since the publication of the report of the Chicago meeting, over 200 members, who had not paid their 1933 dues at that time, have remitted their dues and received their 1933 membership cards. As a result, the number of paid-up members has risen over the 3,300 mark. In other words, approximately 80 per cent of the membership have their dues paid in full. About 75 members who have been unable to pay their dues have promised to do so as soon as possible. Eighteen members have already paid their 1934 dues, although the notices have not yet been mailed.

Recently we had occasion to refer to the annual report made at the 1923 meeting. It was interesting to note how the years 1933 and 1923 compared, as far as the payment of dues was concerned. Here are the figures taken from the reports of the two years.

Year	Paid in full	D-1	D-2
1923	2,995	569	403
1933	3,093	589	407

The designation D-1 means that dues for one year were unpaid. D-2 means that dues for two years were unpaid. Note how closely the figures for 1923 and 1933 compare. Keep in mind that these figures were compiled for the annual reports of these years, presented at the annual meetings. They do not show final figures for these years.

Glancing through the reports for the year between 1923 and 1933, it is noted that in no year did the number of paid-up members reach the 4,000 mark, although in both 1929 and 1930 the

number exceeded 3,900. Everything considered, the members of the A. V. M. A. have met their obligations unusually well, if we may judge from reports made by other organizations. And it should be added that no high-pressure methods have been employed to collect the dues this year, either. Scores of letters have been received from members who were a bit slow in remitting. Each one of them has had a story. Human interest—and lots of it.

In the Secretary's report, made at Chicago, Hawaii was charged with one delinquent member. A letter just recently received indicates that this member was dead when the report was compiled, although this information did not reach the office until the past month. Hawaii should have been credited with 16 paid-up members and no delinquents, and should have had an honor star in table II on page 481 of the October issue of the JOURNAL.

PRESIDENT FITCH INSPECTS OFFICE

Dr. C. P. Fitch, president of the American Veterinary Medical Association, made an inspection of the new A. V. M. A. office while in Chicago, on November 22, en route to Atlanta, Ga., to attend the annual meeting of the Southern States Veterinary Medical Association, November 23 and 24.

EXECUTIVE BOARD ELECTION

The special primary election held in Executive Board District 5, for the purpose of choosing a successor to Dr. C. P. Fitch, following his election to the presidency, came to a close November 13, November 12 having been a Sunday. As has happened so frequently in primary elections held during recent years, there was a tie for fifth place, which made it necessary for the Election Committee to certify to more than the statutory number (five) of nominees prescribed by the By-laws. The tie was a three-cornered one, and this necessitated placing seven names on the ballot. Five of the nominees are from Iowa and two from Minnesota. Three are practitioners, three are college men and the seventh is a B. A. I. veterinarian. Ballots for the regular election have been mailed to every paid-up member in Iowa and Minnesota. The polls will remain open for the customary sixty days, which means that members will have an opportunity to vote until January 13, 1934. Following are the nominees, with brief outlines of their A. V. M. A. activities:

BARGER, J. A. — Des Moines, Iowa

Inspector-in-charge, U. S. Bureau of Animal Industry. Graduate of Kansas City Veterinary College, 1908. Joined A. V. M. A., 1919. Fourth vice-president, 1929-30.

BERGMAN, H. D. — Ames, Iowa

Head, Veterinary Physiology and Pharmacology, Iowa State College. Graduate of Iowa State College, 1910. Joined A. V. M. A., 1913. Resident secretary for Iowa, 1917-18, 1919-21; member (1918-21; 1922-23) and chairman (1921-22) of Committee on Intelligence and Education; member of Committee on Unofficial Veterinary Remedies 1919-20; member of Committee on Local Arrangements, 1923-24; chairman of Committee on Proprietary Pharmaceuticals, 1927-.

COVAULT, C. H. — Ames, Iowa

Professor of Veterinary Medicine, Iowa State College. Graduate of Ohio State University, 1911. Joined A. V. M. A., 1918.

KERNKAMP, H. C. H. — Saint Paul, Minn.

Associate professor of Veterinary Medicine, University of Minnesota. Graduate of Ohio State University, 1914. Joined A. V. M. A., 1916. Chairman of Section on Education and Research, 1923-24; resident secretary for Minnesota, 1924-27; 1928-31.

PARKER, F. F. — Des Moines, Iowa

Practitioner. Graduate of Iowa State College, 1900. Joined A. V. M. A., 1924. Chairman of Section on Small Animals, 1930-31.

SCOTT, C. J. — Knoxville, Iowa

Practitioner. Graduate of Iowa State College, 1908. Joined A. V. M. A., 1909. Member of Special Committee on Affiliation of State and Provincial Associations with the A. V. M. A., 1929-32; member of Committee on Resolutions, 1930-32.

WEST, R. L. — Waseca, Minn.

Practitioner. Graduate of Kansas City Veterinary College, 1918. Joined A. V. M. A., 1919. Member of Executive Board, 1933-.

APPLICATIONS FOR MEMBERSHIP

(See July, 1933, JOURNAL)

FIRST LISTING

MCGINNIS, C. L. — Far Hills, N. J.

D. V. M., Kansas State College, 1933
Vouchers: R. R. Dykstra and M. L. Morris.

Applications Pending

SECOND LISTING

(See November, 1933, JOURNAL)

Apt, Samuel, 221 S. Cecil St., Philadelphia, Pa.
Brown, Victor R., Ontario Veterinary College, Guelph, Ont., Can.
Hampton, Willard, Buffalo, Wyo.
Royer, Harry K., Stevens, Pa.

The amount which should accompany an application filed this month is $5.42, which covers membership fee and dues to January 1, 1934, including subscription to the JOURNAL. It is suggested that applications filed this month be accompanied by remittance for $10.42, the additional $5.00 being for the 1934 dues.

COMING VETERINARY MEETINGS

Horse Association of America. Blackstone Hotel, Chicago, Ill. December 6, 1933. Mr. Wayne Dinsmore, Secretary, Union Stock Yards, Chicago, Ill.

New York City, Veterinary Medical Association of. Academy of Medicine, 5th Ave. and 103rd St., New York, N. Y. December 6, 1933. Dr. John E. Crawford, Secretary, 708 Beach & 19th Sts., Far Rockaway, Long Island, N. Y.

United States Live Stock Sanitary Association. Hotel La Salle, Chicago, Ill. December 6-8, 1933. Dr. O. E. Dyson, Secretary, 45 Live Stock Exchange, Wichita, Kan.

East Tennessee Veterinary Medical Society. White Surgical Supply Co., Knoxville, Tenn. December 9, 1933. Dr. R. E. Baker, Secretary, Morristown, Tenn.

Interstate Veterinary Medical Association. Elks Building, Omaha, Neb. December 11, 1933. Dr. G. L. Taylor, Secretary, Plattsmouth, Neb.

San Diego County Veterinary Medical Association. San Diego, Calif. December 12, 1933. Dr. L. K. Knighton, Secretary, 3438 Mountain View, San Diego, Calif.

Chicago Veterinary Medical Society. Hotel La Salle, Chicago, Ill. December 12, 1933. Dr. O. Norling-Christensen, Secretary, 1904 W. North Ave., Chicago, Ill.

Nebraska State Veterinary Medical Association. Hotel Lincoln, Lincoln, Neb. December 12-13, 1933. Dr. E. C. Jones, Secretary, c/o Platte Valley Serum Co., Grand Island, Neb.

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. December 13, 1933. Dr. A. S. Schlingman, Secretary, Parke, Davis & Co., Detroit, Mich.

Northern Tri-State Veterinary Association. Androy Hotel, Superior, Wis. December 14, 1933. Dr. Robert Dixon, Secretary, 36 Laurel Ave., Superior, Wis.

South Texas Veterinary Medical Association. Plaza Hotel, Laredo, Tex. December 14, 1933. Dr. J. K. Northway, Secretary, Kingsville, Tex.

Tulsa County Veterinary Association. Tulsa, Okla. December 14, 1933. Dr. J. M. Higgins, Secretary, 3305 E. 11th St., Tulsa, Okla.

Western New York Veterinary Medical Association. Buffalo, N. Y. December 14, 1933. Dr. F. F. Fehr, Secretary, 243 S. Elmwood Ave., Buffalo, N. Y.

South Dakota Veterinary Medical Association. Hotel Cataract, Sioux Falls, S. Dak. December 14-15, 1933. Dr. Geo. E. Melody, Secretary, Hoven, S. Dak.

Kansas City Veterinary Association. Baltimore Hotel, Kansas City, Mo. December 19, 1933. Dr. J. D. Ray, Secretary, 1103 E. 47th St., Kansas City, Mo.

Southern California Veterinary Medical Association. Chamber of Commerce Building, Los Angeles, Calif. December 20, 1933. Dr. T. G. Beard, Secretary, 3684 Beverly Blvd., Los Angeles, Calif.

Keystone Veterinary Medical Association. Philadelphia, Pa. December 27, 1933. Dr. C. S. Rockwell, Secretary, 5225 Spruce St., Philadelphia, Pa.

American Association for the Advancement of Science. Boston, Mass. December 27, 1933-January 2, 1934. Dr. Henry B. Ward, Secretary, Smithsonian Institution Bldg., Washington, D. C.

California State Veterinary Medical Association and University of California Veterinary Conference. University Farm, Davis, Calif. January 2-5, 1934. Dr. Geo. M. Simmons, Secretary, 1386 Golden Gate Ave., San Francisco, Calif.

Western Michigan Veterinary Medical Association. Grand Rapids, Mich. January 4, 1934. Dr. C. H. Haasjes, Secretary, 728 S. State St., Shelby, Mich.

New Jersey Veterinary Medical Association of. Hotel Douglas, Newark, N. J. January 4-5, 1934. Dr. John G. Hardenbergh, Secretary, c/o Walker-Gordon Lab. Co., Plainsboro, N. J.

Oklahoma Veterinary Medical Association. Biltmore Hotel, Oklahoma City, Okla. January 8-9, 1934. Dr. C. H. Fauks, Secretary, 1719 S. W. 15th St., Oklahoma City, Okla.

Intermountain Livestock Sanitary Association. Ogden, Utah. January 8-10, 1934. Dr. D. E. Madsen, Secretary, Utah Experiment Station, Logan, Utah.

Pennsylvania, Conference for Veterinarians at University of. School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa. January 9-10, 1934. Dr. G. A. Dick, Dean, 39th St. and Woodland Ave., Philadelphia, Pa.

Maine Veterinary Medical Association. State House, Augusta, Me. January 10, 1934. Dr. L. E. Maddocks, Secretary, R. F. D. 2, Augusta, Me.

Cornell University, Annual Conference for Veterinarians at. New York State Veterinary College, Ithaca, N. Y. January 11-12, 1934. Dr. W. A. Hagan, Dean, Cornell University, Ithaca, N. Y.

Texas, State Veterinary Medical Association of. Kyle Hotel, Temple, Tex. January 15-16, 1934. Dr. D. Pearce, Secretary, Box 335, Leonard, Tex.

Indiana Veterinary Medical Association. Severin Hotel, Indianapolis, Ind. January 16-18, 1934. Dr. W. B. Craig, Secretary, 1420 N. Alabama St., Indianapolis, Ind.

Kansas Veterinary Medical Association and Kansas State College Conference for Veterinarians. Kansas State College, Manhattan, Kan. January 17-18, 1934. Dr. Chas. W. Bower, Secretary, 1128 Kansas Ave., Topeka, Kan.

Vermont Veterinary Medical Association. Montpelier Tavern, Montpelier, Vt. January 19, 1934. Dr. G. N. Welch, Secretary, 43 Union St., Northfield, Vt.

South Carolina Association of Veterinarians. Jefferson Hotel, Columbia, S. C. January 22, 1934. Dr. G. J. Lawhon, Secretary, Hartsville, S. C.

Wisconsin Veterinary Medical Association. Park Hotel, Madison, Wis. January 22-24, 1934. Dr. B. A. Beach, Secretary, University of Wisconsin, Madison, Wis.

Michigan State College Short Course for Veterinarians. Michigan State College, East Lansing, Mich. January 22-26, 1934, Dr. Ward Giltner, Dean, Michigan State College, East Lansing, Mich.

Iowa Veterinary Medical Association. Hotel Savery, Des Moines, Iowa. January 23-25, 1934. Dr. C. J. Scott, Secretary, Knoxville, Iowa.

Massachusetts Veterinary Association. Hotel Westminster, Boston, Mass. January 24, 1934 (annual). Dr. H. W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.

Minnesota State Veterinary Medical Society. Saint Francis Hotel, Saint Paul, Minn. January 25-26, 1934. Dr. C. P. Fitch, Secretary, University Farm, Saint Paul, Minn.

Ohio State Veterinary Medical Association. Neil House, Columbus, Ohio. January 25-26, 1934. Dr. R. E. Rebrassier, Secretary, Ohio State University, Columbus, Ohio.

Nevada State Veterinary Association. University of Nevada, Reno, Nev. January 30, 1934. Dr. Warren B. Earl, Secretary, Box 1027, Reno, Nev.

Missouri Veterinary Medical Association and Special Course for Graduate Veterinarians. University of Missouri, Columbia, Mo. January 30-31, February 1, 1934. Dr. Ashe Lockhart, Secretary, 800 Woodswether Rd., Kansas City, Mo.

FURTHER STUDIES ON NATURAL BRUCELLA INFECTION IN SWINE*

By HOWARD W. JOHNSON, I. FOREST HUDDLESON *and* E. E. HAMANN

Central Brucella Station, Bacteriological Section
Michigan State Experiment Station
East Lansing, Mich.

The discovery by Traum[1] and by Good[2] of Brucella infection in the hog and the association of infection with premature expulsion of fetuses, focused subsequent studies chiefly on the abortion phase of the disease and experimental attempts to produce this symptom. For a time, the appearance of abortions was considered the main criterion of the presence of the disease in a herd of hogs.

In more recent years there have appeared data which show conclusively that the symptom, abortion, can not be depended upon as an indication of the presence or absence of Brucella infection in a herd of hogs. In other words, there may occur abortions in a herd of hogs not infected with Brucella, or there may be infection without the occurrence of abortions. This point is emphasized in a study of the incidence of brucellosis in hogs on farms in Iowa, by Murray and McNutt.[3] They found no record of abortions occurring in 13 of 21 herds of hogs containing positive agglutination reactors. In one of three herds containing no positive reacting animals, abortions had been observed.

In a recent study of natural Brucella infection in a herd of swine in California, Howarth and Hayes[4] report the occurrence of five abortions among 31 pregnant reacting sows. The percentage of pig crop and the number of live pigs born were just as large in the reacting sows as in those that were consistently negative to the agglutination test. They were unable to recover Brucella from uterine swabs, dead fetuses and placentas except in a few cases. In a study of one large herd reported by us,[5] it was pointed out that during two consecutive farrowing seasons, only one sow aborted as a result of *Brucella suis* infection. When abortion occurs in swine, the only certain evidence that they are due to Brucella is the recovery of the organism in cul-

*Journal Article No. 111, New Series, from the Michigan Agricultural experiment Station. Presented at the sixty-ninth annual meeting of the American Veterinary Medical Association, Atlanta, Ga., August 23-26, 1932. This study was financed partly by a grant from the Abortion Committee of the National Research Council.

ture from fetal tissues, regardless of whether the aborting sows show agglutinins in their blood.

The importance of other bacteria in the causation of the abortions in pregnant sows and loss of pigs at farrowing time, even though they show evidence of Brucella infection by the presence of agglutinins in their blood, was pointed out by Graham, Boughton and Tunnicliff[6] and by the writers.[5] Other symptoms of Brucella infection in the hog which may prove to be important are those of lameness and stiffness described by James and Graham.[7] The lesions which gave rise to these symptoms were pyemic arthritis, pyemic bursitis and osteomyelitis. *Br. suis* was recovered from the lesions.

The studies of natural Brucella infection in swine in one herd, which were reported on by us, have been continued and extended to two other large herds. The data presented in this paper concern the epizoölogy of the disease in its various aspects in each of the three herds while they were maintained under different systems of management.

Method of Study

Serological examinations: When blood specimens from the hogs were desired, tail-bleeding was resorted to. Sufficient blood for examination may be collected in this way in a very short time. The rapid agglutination method for detecting Brucella agglutinins in serum was used throughout the study. Occasionally it was compared with the test-tube method for accuracy. The results obtained by the two methods were always in close agreement. The amounts of serum used in titrating each blood specimen for agglutinins were 0.08, 0.04, 0.02, 0.01 and 0.004 cc. These respective amounts of serum represent dilutions of 1:25, 1:50, 1:100, 1:200 and 1:500, in the test-tube test, when used with 2 cc of antigen. The following symbols indicate the reactions obtained:—, negative; T, trace of agglutination; P, incomplete agglutination; +, complete agglutination. In the tables where the dilution figures are not placed at the top of the columns containing the serological reactions, the amounts of serum used are the same as those just mentioned.

Bacteriological examinations: When a hog was slaughtered, pieces of tissue were taken from each of the internal organs. Pieces of the muscles and skeletal and visceral lymph-nodes also were removed from different parts of the carcass. The cut surfaces of the pieces of tissue were smeared over the surface of several beef-liver-agar plates containing gentian violet in a 1:200,000 dilution. The inoculated medium then was incubated

both aerobically and in an increased atmosphere of CO_2 at 37°C. for seven days before being discarded. All cultures isolated were identified by means of the agglutination test, hydrogen sulfid metabolism and the dye media differential method.[8]

Results of Study

Each of the three herds in which the studies were conducted contained from 180 to 260 young and mature hogs. While many were slaughtered during the year, a large number of the best types were kept for breeding purposes.

Herd A

Herd A came under our observation in January, 1930. The herd contained the following breeds: Yorkshire, Berkshire, Hampshire, Chester White, Duroc, Poland China and Tamworth.

The sanitary condition under which the herd was maintained was better than one usually finds on the average hog farm. During the winter, they were quartered inside in small pens. In the summer they were kept in feed-lots. At the time of farrowing, each sow was placed in an enclosure separate from the other hogs.

A study of the records of the herd revealed no loss from premature expulsion of fetuses of any consequence. There were, however, records of the occurrence in the past of the delivery of many still-born pigs at the time of farrowing. Sterility in brood sows had been observed.

The first agglutination test conducted on this herd in 1930 revealed 18 hogs reacting in a titre of 1:100 or above and 28 in titres below 1:100. In other words, the test furnished evidence that 46 animals (25.5 per cent) in the herd had been exposed to Brucella. Criticism might be raised of the interpretation of reactions as low as a trace in a 1:25 dilution as meaning exposure to Brucella infection, but subsequent tests and bacteriological findings revealed that low reactions in many instances have significance.

In herd A, an effort first was put forth to control the spread of the disease by blood-testing each animal in the entire herd at intervals of 30 days and segregating those showing agglutinins in a titre of 1:50 or higher from the remaining herd. The high-titre ones were slaughtered as quickly as possible. We were not long in finding a defect in this procedure. Many of the animals that were left in the negative group, and showing only an incomplete agglutination reaction in a titre of 1:25, were found on a succeeding 30-day test to react in a high titre.

By removing all animals showing any degree of agglutinins in their blood, the further progress of the disease in the herd was checked. In addition, all animals purchased for replacement were examined for Brucella agglutinins before they were added to the negative herd. The farrowing pens were cleaned thoroughly and disinfected. After farrowing, the sows and pigs were placed on ground which had never been used by hogs. As a result of this procedure, a herd of negative animals was obtained in less than eight months from the time the study was undertaken.

One of the surprising results of the study was to find the agglutination titres, of the entire group of reacting animals not slaughtered, decrease to the vanishing point. The trend of the agglutination titres in these animals is set forth in table I. This group was made up of 23 sows, gilts and boars of seven different breeds. Within seven months after the testing was begun, all, with the exception of two animals, were negative to the agglutination test. On subsequent tests these two also became negative and the remaining ones remained negative. The spring crop of pigs was placed on test in the fall and the fall crop during the winter months of 1931. The test was now carried out at 60-day intervals. Those animals remaining in the herd which had become negative were placed with the negative animals during the fall of 1930.

For the purpose of clarity, the incidence of infection in herd A under this plan of controlling the disease is set forth in graphs (figs. 1 and 2). The incidence of infection is based on the percentage of hogs reacting in a titre of 1:50 or above out of the total number in the herd at the time each test was made.

HERD B

A study of the disease in herd B was begun in May, 1930. The herd consisted of 132 Duroc hogs. The first agglutination test revealed 72 animals reacting in a titre of 1:50 and above.

During the course of the study of the disease in herd B, no attempt was made to prevent the spread of the disease in the older hogs. The young pigs, after weaning, were kept separate from the older hogs until they reached maturity. Each month, from 20 to 30 of the older animals were slaughtered at the farm for pork. No attempt was made to select animals for slaughter on the basis of the agglutination reaction. It appears from the curve of the incidence of infection shown in figures 1 and 2 that the spread of the disease in herd B not only was checked but was considerably reduced by the elimination of many infected animals

at the time of slaughter and by maintaining the young ones in quarters separate from older ones. Soon after the young animals were placed with older ones, a rapid rise in the incidence of infection took place. This is shown clearly by the curve in figure I, where only newly infected animals are considered.

Herd C

The study of the disease in herd C was begun in June, 1930. The herd consisted of 447 Duroc hogs. The first agglutination test revealed 107 animals reaction in a titre of 1:50 or above. There was little if any history of premature expulsion of fetuses in the herd. Although several sows had failed to conceive in the past, the number involved was never sufficient to cause alarm.

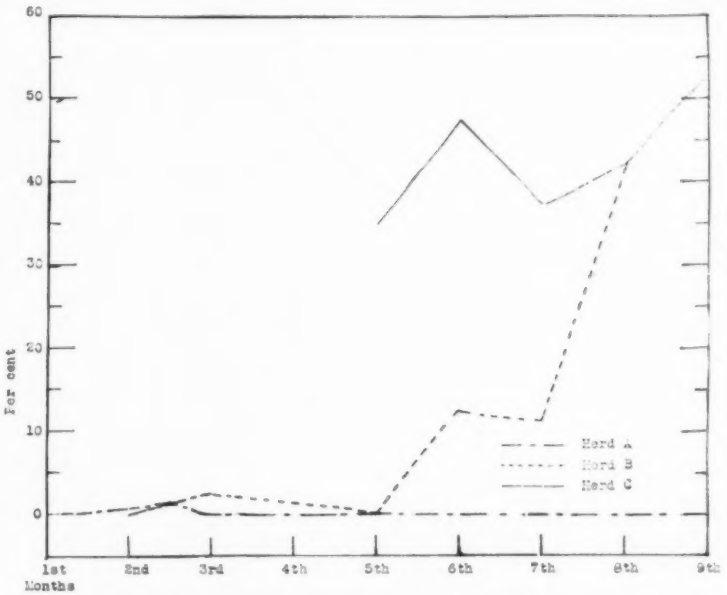

Fig. 1. Graph showing the incidence of new Brucella reactions in three herds of swine.

During the months that followed after the first blood-test, no precaution whatever was taken to prevent the spread of disease from the infected to the non-infected. Even the younger pigs were not kept in quarters which would prevent infection from reaching them from older infected ones. Many of the older animals were slaughtered at the farm at wide intervals, but no attention was given to their blood-reaction. The course of the

disease in this herd is illustrated by the shape of the curves in figures 1 and 2. It is particularly interesting to compare the curve of the incidence of infection in this herd with those of herds A and B. Although many infected animals were removed from the herd by slaughter, and new offspring were added from time to time, the incidence of infection continued to increase. The increase in the incidence of infection is due possibly to the maintenance of offspring in infected quarters and association with infected older animals shortly after weaning. It is of interest to note that, during the period in which this herd was under our observation (over one year), not a single abortion occurred.

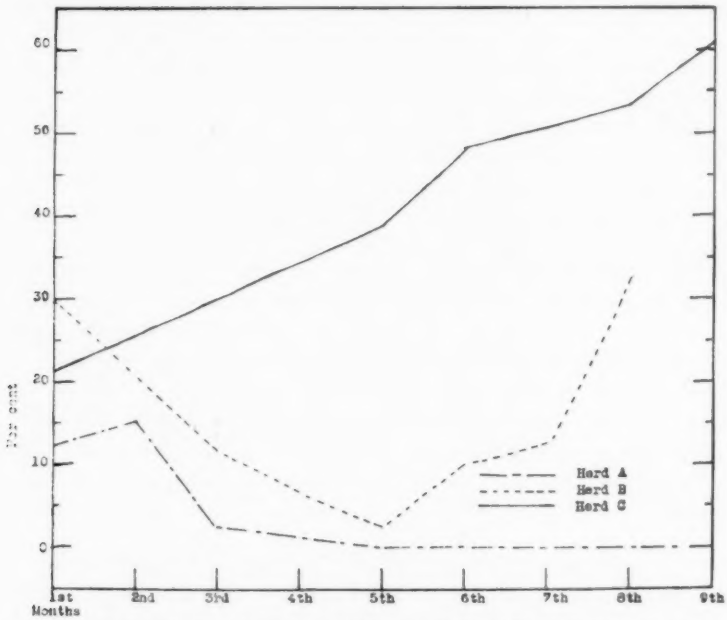

FIG. 2. Graph showing the incidence of Brucella infection in three herds of hogs maintained under different methods of control.

In analyzing the trend of the agglutination titre in 123 unslaughtered hogs in herds B and C for a five-month period, it was found that of those reacting in a titre as high as a 1:100, 75 (60 per cent) showed either no decrease or an increase in agglutination titre. The remaining 48 animals showed either a marked decrease in titre or never reacted as high as a 1:100 titre during the five-month period. These results are quite contradictory to those recorded in our study of herd A (table I),

TABLE I—*Trend of agglutination titres in segregated reacting hogs.*

1930			JANUARY	FEBRUARY	APRIL	JUNE	JULY	AUGUST	SEPTEMBER
			AGGLUTINATION TITRES						
HOG	BREED	SEX	1:25 1:50 1:100 1:200 1:500	1:25 1:50 1:100 1:200 1:500	1:25 1:50 1:100 1:200 1:500	1:25 1:50 1:100 1:200 1:500	1:25 1:50 1:100 1:200 1:500	1:25 1:50 1:100 1:200 1:500	1:25 1:50 1:100 1:200 1:500
1	Poland China	Boar	++ P T−	+++ P T	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −
2	Duroc	Boar	T− − − −	P T− − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −
3	Yorkshire	Sow	P T− − −	− − − − −	P T− − −	− − − − −	+ P T− −	− − − − −	− − − − −
4	Duroc	Sow	− − − − −	P T− − −	T− − − −	P T− − −	− − − − −	− − − − −	− − − − −
5	Poland China	Gilt	− − − − −	P T− − −	T− − − −	− − − − −	− − − − −	− − − − −	− − − − −
6	Poland China	Gilt	− − − − −	− − − − −	T− − − −	+ P T− −	T− − − −	− − − − −	− − − − −
7	Yorkshire	Sow	T− − − −	P T− − −	P T− − −	+ P T− −	T− − − −	P T− − −	P T− − −
8	Chester	Gilt	− − − − −	P T− − −	P T− − −	P T− − −	P T− − −	− − − − −	− − − − −
9	Hampshire	Sow	P T− − −	P T− − −	P T− − −	T− − − −	P T− − −	P T− − −	P T− − −
10	Yorkshire	Sow	P P T− −	+ P T− −	P P T− −	P P T− −	P P T− −		
11	Poland China	Sow	− − − − −	+ P T− −	T− − − −	− − − − −	P T− − −		
12	Yorkshire	Sow	− − − − −	− − − − −	P T− − −	− − − − −	− − − − −	− − − − −	− − − − −
13	Chester	Sow	++ P T−	P P T− −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −
14	Chester	Gilt	+++++ P	+++++ P	P T− − −	− − − − −	T− − − −	− − − − −	− − − − −
15	Berkshire	Sow	+++++ P	++ P T−	P T− − −	P P T− −	− − − − −	− − − − −	− − − − −
16	Yorkshire	Boar	P T− − −	− − − − −	− − − − −	T− − − −	T− − − −	T− − − −	− − − − −
17	Tamworth	Sow	T− − − −	+ P T− −	T− − − −	− − − − −	− − − − −	− − − − −	− − − − −
18	Duroc	Gilt	+++ P T	++ P T−	T− − − −	− − − − −	− − − − −	− − − − −	− − − − −
19	Berkshire	Gilt	+++ P T	+++++ P	++++++	P P T− −	− − − − −		
20	Berkshire	Gilt	++++++	++++++	++++++	P P T− −	− − − − −		
21	Duroc	Boar		P T− − −	P T− − −	++++++	+++ P T	P T− − −	− − − − −
22	Chester	Boar		++ P T−	+ P P T−	+ P T− −	P P T− −	− − − − −	− − − − −
23	Berkshire	Boar	P P T− −	++ P T−	++ P T−	++ P T−	++ P T−	T− − − −	− − − − −

in which all reacting animals, regardless of maximum titre, became negative.

Location of Brucella Suis in Infected Hogs

During the period of study of the trend of the agglutination titres in the three herds of hogs, many of them were slaughtered for meat. From these, the internal organs were obtained for cultural study and at the same time a specimen of blood was taken for the agglutination test. The results of this study are compiled in tables II and III. In table II are presented the reactions of 132 animals from which *Br. suis* was not recovered at the time of slaughter. Of these, 53 (39 per cent) were reacting in a titre of 1:100 or higher, at the time of slaughter. The remaining 79 animals either failed to reach a titre of 1:100 or had fallen below this titre when a sample of blood was examined at the time of slaughter. In addition to the 132 reacting animals with negative cultural findings, there were examined bacteriologically 118 slaughtered animals that had always been negative to the agglutination test. The cultural findings on these also were negative.

In table III is recorded the trend of the agglutination titres of 46 hogs in the three herds in which *Br. suis* was recovered from one or more of the organs at the time of slaughter. Although most of the animals from which *Br. suis* was recovered were reacting in a 1:500 titre at the time of slaughter, it is interesting to note that the cultural findings were positive in several animals showing a very low agglutination titre. Two animals in particular (40 and 45) showed only an incomplete reaction in a 1:25 dilution at the time they were slaughtered. The organism was recovered from the spleen of one and from the liver, spleen and mesenteric lymph-nodes of the other.

The bacteriological findings, as summarized in table IV, on 46 naturally infected animals from the three herds, furnish evidence of the chief location of *Br. suis* in the hog and the probable nature of the disease. The organism is likely to be found in the spleen more often than in any other one organ. The other organs of the hog, however, should not be neglected in searching for *Br. suis*, for occasionally the organism was found in other organs when it appeared to be absent in the spleen. The predilection of the organism for the tissues named in table IV would indicate that the disease is of the nature of a splenic lymphadenitis, and the occurrence of abortion in a pregnant female is of secondary or purely incidental significance.

TABLE II—*Agglutination reactions of hogs not showing Brucella suis at time of slaughter.*

HOG	BREED	SEX	MONTHLY AGGLUTINATION TITRES					INTERVAL BETWEEN 5TH MONTHLY TEST AND TIME OF SLAUGHTER (DAYS)	SERUM TITRE AT TIME OF SLAUGHTER
			1ST	2ND	3RD	4TH	5TH		
1	Duroc	Sow	− − − − −	+ + + + +	+ + + + +	+ + + + +	+ + + + +	20	+ + + + +
2	Duroc	Gilt	+ + + P T	+ + + + P	+ + + + +	+ + + + +	+ + + + +	19	+ + + + P
3	Duroc	Gilt	+ + + P T	+ + + P T	+ + + + +	+ + + + +	+ + + + +	19	+ + + + +
4	Duroc	Gilt	− − − − −	+ + + + +	+ + + + +	+ + + + +	+ + + + +	19	+ + + + +
5	Duroc	Gilt	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	19	+ + + + +
6	Duroc	Barrow	P T − − −	+ + + P T	+ + + + P	+ + + + +	+ + + + +	19	+ + + + +
7	Duroc	Barrow	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	20	+ + + + +
8	Duroc	Barrow	P P T − −	+ + P T −	+ + + + +	+ + + + P	+ + + + +	19	+ + + + +
9	Duroc	Gilt	+ + + + +	+ + + + +	+ + + + +	+ + + P T	+ + + + +	19	+ + + + +
10	Duroc	Barrow	− − − − −	+ + + P T	+ + + + +	+ + + + +	+ + + + +	22	+ + + + +
11	Duroc	Gilt	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	20	+ + + + +
12	Duroc	Barrow	− − − − −	− − − − −	− − − − −	− − − − −	+ + + + +	126	+ + + + +
13	Duroc	Gilt	− − − − −	+ + + P T	+ + + + +	+ + + + +		98	+ + + P T
14	Duroc	Sow	P T − − −	+ + + + +	+ + + + +	+ + + + +		98	+ P T − −
15	Duroc	Sow	+ + + + +	+ + + + +	+ + + + +			98	+ + + P T
16	Duroc	Gilt	+ + + + +	+ + P T −	+ + + + +	+ + + P T	+ + + + +	20	+ + + + +
17	Duroc	Barrow	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	20	+ + + + +
18	Duroc	Sow	+ + + + +	T − − − −	− − − − −	+ + P T −	+ + + + +	59	+ + + + +
19	Duroc	Barrow	− − − − −	− − − − −	+ + + P T	+ + + + +	+ + + + +	19	+ + + + +
20	Duroc	Barrow	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	58	+ + + + +
21	Duroc	Barrow	+ + + + +	+ + P T −	+ + + + +	+ + + + +		59	+ + + + +
22	Duroc	Barrow	T − − − −	− − − − −	+ + + + +	+ + + + +	+ + + + +	58	+ + + + +
23	Duroc	Barrow	+ + + + +	+ + + + +	+ + + + +			58	+ + + + +

TABLE II—*Agglutination reactions of hogs not showing Brucella suis at time of slaughter—Continued.*

HOG	BREED	SEX	MONTHLY AGGLUTINATION TITRES					INTERVAL BETWEEN 5TH MONTHLY TEST AND TIME OF SLAUGHTER (DAYS)	SERUM TITRE AT TIME OF SLAUGHTER
			1ST	2ND	3RD	4TH	5TH		
24	Duroc	Sow	+ P T − −	+ + + P T	+ + + + +			8	+ + + + +
25	Duroc	Gilt	+ + + + +					0	+ + + + +
26	Duroc	Barrow	+ + + + +	+ + + + +	+ + + + P	+ + + + +	+ + + + +	29	+ + + + +
27	Duroc	Barrow	− − − − −	− − − − −	+ + + + +			10	+ + + + +
28	Duroc	Barrow	− − − − −	− − − − −	+ + + + +			10	+ + + P T
29	Duroc	Gilt	+ + + + +					0	+ + + + +
30	Duroc	Barrow	+ + + + +					0	+ + + + +
31	Duroc	Barrow	+ + + + +					0	+ + + + +
32	Duroc	Gilt	+ + + + +					0	+ + + + +
33	Duroc	Gilt	+ + + + +					0	+ + + + +
34	Duroc	Barrow	+ + + + +					0	+ + + + +
35	Duroc	Gilt	+ + + + +					0	+ + + + +
36	Duroc	Barrow	+ + + + +					0	+ + + + +
37	Duroc	Barrow	+ + + + +					0	+ + + + +
38	Duroc	Barrow	+ + + + +					0	+ + + + +
39	Duroc	Gilt	+ + + + +					0	+ + + + +
40	Tamworth	Sow	+ + P T −	+ + + + +	+ + + + P			31	+ P T − −
41	Duroc	Gilt	P P T − −	+ + P T −	+ + + + +	+ + + + +	+ + + + P	19	+ + + + +
42	Duroc	Barrow	+ + + + +	+ + + + +	+ + + + +	+ + + + P	+ + + + P	19	+ + + + P
43	Duroc	Barrow	+ + P T −	+ + + + +	+ + + + +	+ + + + P		96	P P T − −
44	Duroc	Gilt	− − − − −	+ P T − −	+ + + + P	+ + + + P		98	+ + P T −
45	Duroc	Barrow	− − − − −	+ + + P T	− − − − −	+ + + + P		69	+ + + + +
46	Duroc	Barrow	+ + P T −	+ + P T −	+ + + + +	+ + + P T	+ + + + P	58	+ + + + +

TABLE II—*Agglutination reactions of hogs not showing Brucella suis at time of slaughter—Continued.*

Hog	Breed	Sex	Monthly Agglutination Titres					Interval Between 5th Monthly Test and Time of Slaughter (Days)	Serum Titre at Time of Slaughter
			1st	2nd	3rd	4th	5th		
47	Yorkshire	Gilt	− − − − −	− − − − −	− − − − −	+ + + P T		19	+ + + + +
48	Duroc	Barrow	− − − − −	− − − − −	+ P T − −	+ + + + P	+ + + P T	19	+ + + + +
49	Duroc	Barrow	+ + + + +	+ + + + +	+ + + + P	+ + + + P	+ + + P T	19	+ + + + +
50	Duroc	Sow	+ + + + +	P T − − −	+ + + P T	+ + + P T		7	+ + + P T
51	Duroc	Sow	+ + P T −	+ + + + +	+ + + P T			8	+ + + P T
52	Duroc	Sow	+ P T − −	+ + P T −	+ + + P T			8	+ + + P T
53	Duroc	Gilt	+ P T − −	+ + + + +	+ + + + +	+ P T − −	P T − − −	43	+ + + P T
54	Duroc	Gilt	− − − − −	+ + + + +	+ + + P T			10	+ + + P T
55	Duroc	Barrow	+ + + P T					0	+ + + P T
56	Chester	Sow	− − − − −	+ + + P T	+ + P T −	+ + P T −		30	+ + + P T
57	Duroc	Sow	+ + + + P	+ + P T −	+ + + P T	+ P T − −	+ + P T −	30	+ + P T −
58	Duroc	Sow	− − − − −	+ + + + +	+ + + + +	+ + + + +	+ + P T −	96	+ + P T −
59	Duroc	Sow	+ + + + P	+ + + + +	+ + P T −	+ + P T −		98	+ + P T −
60	Duroc	Barrow	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + P T −	59	+ + P T −
61	Duroc	Barrow	− − − − −	− − − − −	− − − − −	− − − − −	+ + P T −	21	+ + + + +
62	Duroc	Gilt	+ P T − −	+ + + + +	+ P T − −	+ P T − −	+ + P T −	59	P P T − −
63	Duroc	Barrow	− − − − −	− − − − −	− − − − −	T − − − −	+ + P T −	58	+ + + P T
64	Duroc	Barrow	+ + + + +	+ + P T −				57	P T − − −
65	Duroc	Sow	+ + P T −	+ + P T −	+ + P T −	+ + P T −		7	+ + P T −
66	Duroc	Barrow	+ + P T −					0	+ + P T −
67	Duroc	Gilt	+ + P T −					0	+ + P T −
68	Duroc	Sow	+ + P T −	+ P P T −	+ P P T −			30	+ P P T −
69	Poland China	Sow	P P T − −	+ P T − −				86	+ P T − −

TABLE II—*Agglutination reactions of hogs not showing Brucella suis at time of slaughter—Continued.*

HOG	BREED	SEX	MONTHLY AGGLUTINATION TITRES					INTERVAL BETWEEN 5TH MONTHLY TEST AND TIME OF SLAUGHTER (DAYS)	SERUM TITRE AT TIME OF SLAUGHTER
			1ST	2ND	3RD	4TH	5TH		
70	Duroc	Sow	− − − − −	P T − − −	+ P T − −			20	P T − − −
71	Duroc	Barrow	+ + P T −	+ P T − −	P T − − −	+ P T − −		146	P P T − −
72	Duroc	Barrow	T − − − −	− − − − −	P P T − −	+ + P T −	+ P T − −	58	+ + + P T
73	Duroc	Sow	− − − − −	− − − − −	T − − − −	+ P T − −		8	− − − − −
74	Duroc	Sow	+ + + P T	+ + + P T	+ + P T −	+ P T − −		7	+ P T − −
75	Duroc	Sow	+ P T − −	+ P T − −				46	+ P T − −
76	Duroc	Gilt	+ P T − −					0	+ P T − −
77	Duroc	Gilt	+ P T − −					8	− − − − −
78	Duroc	Gilt	+ + + + +	+ + P T −	+ P T − −			38	+ P T − −
79	Duroc	Barrow	+ P T − −					0	+ P T − −
80	Duroc	Barrow	− − − − −	− − − − −	P T − − −	+ P T − −		10	P T − − −
81	Duroc	Gilt	− − − − −	− − − − −	+ P T − −			10	T − − − −
82	Duroc	Gilt	P P T − −	+ P T − −				30	+ P T − −
83	Duroc	Sow	− − − − −	P T − − −	P T − − −	− − − − −	P T − − −	19	P T − − −
84	Tamworth	Sow	− − − − −	+ P T − −	P T − − −	P T − − −		20	P T − − −
85	Yorkshire	Sow	P P T − −	+ P T − −	P P T − −	P P T − −		20	P P T − −
86	Poland China	Sow	− − − − −	+ + + + P	P T − − −	P T − − −		20	P T − − −
87	Berkshire	Sow	+ P T − −	P T − − −	P P T − −	P T − − −		20	− − − − −
88	Berkshire	Gilt	+ + + P T	+ + + + P	+ + + + +	P P T − −		84	− − − − −
89	Berkshire	Gilt	+ + + + +	+ + + + +	+ + + + +	P P T − −		20	− − − − −
90	Duroc	Barrow	+ + + + P	+ + + P T	P P T − −	P T − − −	P T − − −	20	− − − − −
91	Duroc	Barrow	P P T − −	+ + P P T	P P T − −	P T − − −	P T − − −	20	P P T − −

TABLE II—*Agglutination reactions of hogs not showing Brucella suis at time of slaughter—Continued.*

HOG	BREED	SEX	MONTHLY AGGLUTINATION TITRES					INTERVAL BETWEEN 5TH MONTHLY TEST AND TIME OF SLAUGHTER (DAYS)	SERUM TITRE AT TIME OF SLAUGHTER
			1ST	2ND	3RD	4TH	5TH		
92	Duroc	Barrow	− − − − −	+ + P T −	+ + P T −	T T − − −		33	T T − − −
93	Duroc	Gilt	P T − − −	P T − − −	P T − − −			146	T − − − −
94	Duroc	Gilt	P T − − −	+ + + P T	+ + P T −	+ P P T −	P P T − −	59	− − − − −
95	Duroc	Sow	− − − − −	− − − − −	P T − − −			8	− − − − −
96	Duroc	Sow	T − − − −	T − − − −	P T − − −			8	− − − − −
97	Duroc	Sow	− − − − −	− − − − −	P P T − −			8	− − − − −
98	Duroc	Sow	− − − − −	P T − − −	P T − − −			8	P T − − −
99	Duroc	Sow	P T − − −	+ + P T −	P T − − −	− − − − −	− − − − −	43	P P T − −
100	Duroc	Gilt	− − − − −	P P T − −				8	P P T − −
101	Duroc	Gilt	P P T − −					0	− − − − −
102	Duroc	Barrow	− − − − −	− − − − −	− − − − −	− − − − −	P T − − −	20	− − − − −
103	Duroc	Barrow	P T − − −	P T − − −	− − − − −			50	− − − − −
104	Duroc	Barrow	− − − − −	− − − − −	T − − − −	P T − − −		20	− − − − −
105	Duroc	Barrow	P T − − −					14	− − − − −
106	Duroc	Gilt	P P T − −					0	P P T − −
107	Duroc	Barrow	P T − − −					0	P T − − −
108	Duroc	Gilt	P T − − −					0	P T − − −
109	Duroc	Gilt	− − − − −	+ + + P T	T − − − −			30	+ + P P T
110	Duroc	Gilt	+ + + + +	+ + + + +	+ P T − −	P T − − −		3	P P T T −
111	Berkshire	Gilt	+ + + + +	+ + + + P				20	+ + P T −
112	Chester	Sow	+ + P T −	+ + + + +	+ + P T −	T − − − −		20	T − − − −
113	Berkshire	Sow	+ + + P T	P T − − −	T − − − −			20	T − − − −
114	Duroc	Sow	− − − − −	P T − − −	+ + + P T	T − − − −		98	P T − − −

TABLE II—*Agglutination reactions of hogs not showing Brucella suis at time of slaughter—Concluded.*

HOG	BREED	SEX	MONTHLY AGGLUTINATION TITRES					INTERVAL BETWEEN 5TH MONTHLY TEST AND TIME OF SLAUGHTER (DAYS)	SERUM TITRE AT TIME OF SLAUGHTER
			1ST	2ND	3RD	4TH	5TH		
115	Duroc	Barrow	P T – – –	– – – – –	– – – – –	– – – – –	T – – – –	59	P P T – –
116	Duroc	Barrow	P T – – –	– – – – –	– – – – –	– – – – –	T – – – –	59	+ + + P T
117	Duroc	Sow	– – – – –	– – – – –	T – – – –			14	T – – – –
118	Duroc	Barrow	– – – – –	– – – – –	– – – – –	T – – – –		14	– – – – –
119	Duroc	Barrow	– – – – –	– – – – –	T – – – –			20	– – – – –
120	Duroc	Gilt	– – – – –	T – – – –				20	– – – – –
121	Duroc	Gilt	T – – – –					20	T – – – –
122	Duroc	Sow	P T – – –	+ + + P T	– – – – –			20	– – – – –
123	Berkshire	Sow	T – – – –	+ + + P T	– – – – –	– – – – –		20	– – – – –
124	Duroc	Gilt	+ + + + +	+ + P T –	+ + + P T	– – – – –	– – – – –	19	+ P T – –
125	Duroc	Gilt	+ + + + +	+ + + + +	+ + + P T	P T – – –	– – – – –	20	– – – – –
126	Duroc	Gilt	+ + P T –	+ + + P T				20	+ + + P T
127	Duroc	Gilt	+ + + P T	P T – – –	– – – – –			20	– – – – –
128	Duroc	Sow	T – – – –	– – – – –	– – – – –			20	– – – – –
129	Duroc	Barrow	– – – – –	+ + + P T	+ P T – –	– – – – –	– – – – –	14	– – – – –
130	Duroc	Gilt	– – – – –	– – – – –	+ + + + +			14	P T – – –
131	Duroc	Gilt	T – – – –	T – – – –	– – – – –			42	P P T – –
132	Tamworth	Sow	+ + + + P	T – – – –	– – – – –			84	– – – – –

TABLE III—*Agglutination reactions of hogs showing Brucella suis at time of slaughter.*

HOG	BREED	SEX	MONTHLY AGGLUTINATION TITRES					INTERVAL BETWEEN 5TH MONTHLY TEST AND TIME OF SLAUGHTER (DAYS)	SERUM TITRE AT TIME OF SLAUGHTER	BACTERIOLOGICAL FINDINGS (LOCATION OF *Br. Suis*)
			1ST	2ND	3RD	4TH	5TH			
1	Yorkshire	Gilt	+ + + + +	+ + + + +	+ P T − −	+ + + + +		29	+ + + + +	S, G, I
2	Poland China	Sow	+ + P T −	+ + + + +				45	+ + + + +	S, G, SM
3	Yorkshire	Sow	+ + + + +					17	+ + + + +	U
4	Duroc	Sow	+ + + + +	+ + + + +	+ + + + +			5	+ + + + +	S, L
5	Duroc	Gilt	− − − − −	+ + + + +	+ + + + +	+ + + + +		6	+ + + + +	I, B, SM
6	Duroc	Sow	+ + + + +	+ + + P T	+ + + + +	+ + + + +		6	+ + + + +	S, G
7	Duroc	Barrow	+ + + + +					9	+ + + + +	S
8	Duroc	Gilt	+ + + + +					9	+ + + + +	L, G
9	Duroc	Barrow	+ + + + +					9	+ + + + +	I
10	Duroc	Barrow	+ + + + +					9	+ + + + +	S, SM
11	Duroc	Gilt	+ + + + +					9	+ + + + +	S, SM
12	Duroc	Sow	+ + + P T	+ + + + +	+ + + + +			6	+ + + + +	L, SM
13	Duroc	Sow	+ + + + +	+ + + + +				6	+ + + + +	G
14	Duroc	Barrow	+ + P T −	+ + + + +	+ + + + +	+ + + + +		76	+ + + + +	S
15	Duroc	Barrow	− − − − −	+ + + + +	+ + + + +	+ + + + +		91	+ + + + +	S
16	Duroc	Gilt	+ P T − −	+ + + + +	+ + + + +	+ + + + +		90	+ + + + +	L, S
17	Duroc	Sow	+ + + + +					20	+ + + + +	S
18	Duroc	Sow	+ + + + +					20	+ + + + +	S, G
19	Duroc	Gilt	− − − − −	T − − − −	+ + + + +	+ + + + +	+ + + + +	20	+ + + + +	S, G
20	Duroc	Gilt	+ + + + +	+ + + + +	+ + + + +			20	+ + + + +	S
21	Duroc	Gilt	+ + + + +					0	+ + + + +	I, SM
22	Berkshire	Sow	+ + + P T					17	+ + + P T	MM, S
23	Yorkshire	Gilt	+ + + P P					62	+ + P T −	S
24	Duroc	Sow	P T − − −	+ + + + +				25	+ + P T −	S, K, SB

TABLE III—*Agglutination reactions of hogs showing Brucella suis at time of slaughter—Concluded.*

HOG	BREED	SEX	MONTHLY AGGLUTINATION TITRES					INTERVAL BETWEEN 5TH MONTHLY TEST AND TIME OF SLAUGHTER (DAYS)	SERUM TITRE AT TIME OF SLAUGHTER	BACTERIOLOGICAL FINDINGS (LOCATION OF *Br. Suis*)
			1ST	2ND	3RD	4TH	5TH			
25	Tamworth	Sow	+ + + + +	+ + + P T	+ + + P T			9	+ + + P T	S
26	Chester	Sow	T − − − −	+ + + P T				14	+ + + P T	S, SB, SM
27	Duroc	Gilt	− − − − −	− − − − −	− − − − −	+ + + P T		11	+ + + P T	L
28	Duroc	Sow	+ + + + +	+ + + + +	+ + + + +	+ + + P T		91	+ + + P T	S, G
29	Duroc	Sow	+ + + P T	+ P T − −				43	+ + + + +	K, G, SM
30	Poland China	Sow	P T − − −	+ + P T −				14	+ + P T −	S
31	Chester	Sow	+ + + + +	+ + P T −	+ + P T −			18	+ + P T −	S
32	Poland China	Sow	+ + + P T					84	+ + + + P	SM
33	Duroc	Barrow	P T − − −	+ + P T −				9	+ + P T −	S
34	Duroc	Barrow	− − − − −	+ + P T −				9	+ + P T −	L
35	Duroc	Barrow	+ + P T −					9	+ + P T −	S
36	Duroc	Barrow	− − − − −	− − − − −	− − − − −	+ + P T −		123	+ + P T −	I
37	Duroc	Barrow	− − − − −	− − − − −	− − − − −	+ + P T −		96	+ + P T −	S, L
38	Duroc	Barrow	+ + + + P	+ + P T −				123	+ + P T −	SM, G
39	Duroc	Barrow	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	21	+ P T − −	G, M, B
40	Hampshire	Sow	P T − − −					7*	P T − − −	S, L
41	Yorkshire	Barrow	− − − − −	+ P T − −				3	+ P T − −	S
42	Chester	Sow	P P T − −	+ P T − −				10	+ P T − −	G
43	Duroc	Barrow	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	20	P P T − −	S, L, K, M, I, SM
44	Duroc	Sow	− − − − −	− − − − −	− − − − −	− − − − −		123	+ + + + P	G, K
45	Duroc	Sow	T − − − −	− − − − −	− − − − −			123	P T − − −	S
46	Duroc	Sow	− − − − −	P P T − −	T − − − −	P T − − −	P T − − −	157	+ + + P T	L

*Died.

Key: B=bronchial lymph-nodes. G=gastric lymph-nodes. I=internal iliac lymph-nodes. K=kidney. L=liver. M=mesenteric lymph-nodes. MM=mammary glands. S=spleen. SB=submaxillary lymph-nodes. SM=supramammary lymph-nodes. U=uterus.

In culturing hog tissue, we were surprised to find that the inoculated medium very often must be incubated aerobically five to six days at 37° C. before colonies of *Br. suis* become visible to the eye. In every instance, primary growth occurred under aerobic conditions.

Although we examined blood specimens from 100 reacting hogs for the presence of *Br. suis* with negative results, we were much surprised at these findings in view of the nature of the disease. The recent investigations of Cotton and Buck,[9] however, go far to explain our failure to find the organism in the blood. Cotton and Buck found that *Br. suis* rarely remains in the bloodstream of hogs for a period longer than 45 days after artificial exposure. Their findings would indicate that the disease in its early stages is of the nature of a septicemia.

TABLE IV—*Location of Brucella suis in tissues of 46 naturally infected hogs.*

ORGAN	HOGS SHOWING POSITIVE CULTURE
Spleen	29
Gastric lymph-node	13
Supramammary lymph-nodes	11
Liver	10
Internal iliac lymph-nodes	6
Kidneys	4
Submaxillary lymph-nodes	2
Bronchial lymph-nodes	2
Mesenteric lymph-nodes	2
Uterus	1
Udder	1

From the material that was examined in the beginning of these studies, there were seen certain gross changes in the organs of the infected hogs which we thought would guide one in making a presumptive diagnosis of infection. Later observations on a larger amount of material have failed to substantiate our former findings. The gross changes observed in the organs of infected hogs were seen as often and to the same extent in non-infected ones.

In considering the cause of the failure to obtain positive cultures from the tissues of such a large number of reacting animals, when the organism was obtained so easily from the 46 others, we were led to consider two possible explanations:

First, it was possible that the organism was located in areas

of the carcass which we failed to culture. Second, the continued reactions in the hogs may have been due to their reëxposure to newly infected animals that were eliminating the organism in their excretions. The latter explanation appeared plausible, since we have plenty of evidence from the first herd studied that reacting animals became negative when placed in isolation. In order to test the latter hypothetical explanation, we selected 21 mature hogs in herd C showing high agglutination titres and placed them in a pen separate from the other hogs. They were bled monthly for a period of approximately five months, slaughtered at the end of this period, and their organs cultured for the presence of *Br. suis*. The results of this study are tabulated in table IV. To our surprise, none of the 21 animals became negative. There was, however, a tendency for the reaction to decrease in 12 animals, returning again to a higher titre on subsequent tests. (See Table V.) It is possible that reëxposure was continuing in this pen of hogs. It is of interest to note that *Br. suis* was not cultured from a single one of these animals at the time of slaughter.

TABLE V—*Serological findings on a group of positive hogs isolated for five months. (Cultural findings negative in all cases.)*

HOG	RESULTS OF AGGLUTINATION TESTS				
	11–17–31	12–12–31	1–14–32	2–19–32	3–21–32*
1011	+ + + + +	+ + + + +	+ + + + P	+ + + + P	+ + + + P
1012	+ + + + +	− − − − −	+ + + P T	+ + + P T	+ + + + P
1013	+ + + + P	+ + + P T	P T T − −	P T − − −	+ + + P T
1014	+ + + + +	+ + + + +	+ + + P T	+ + + P T	+ + + + +
1015	+ + + + +	+ + + P T	P P P P T	+ P P P T	+ + + + P
1016	+ + + + +	+ + + + +	+ + + P P	+ + + P T	+ + + + P
1017	+ + + P T	+ + + + P	+ + + + +	+ + + + +	+ + + + +
1018	+ + + + +	+ + + P T	P T − − −	P T − − −	+ + + T −
1019	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +
1020	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + P P
1021	+ + + + +	+ + + + P	+ + + + P	+ + + + +	+ + + + P
1023	+ + + P T	− − − − −	+ + P T −	+ + P T −	+ + + + +
1024	+ + + + +	+ + + P T	+ + P T −	+ + + + +	+ + + P T
1092	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +
1093	+ + + + +	+ + P T −	+ + + + +	+ + + + +	+ + + + +
1094	+ + + + +	+ + + + +	+ + + P T	+ + P T −	+ + P T T
1095	+ + + P T	+ + + + P	+ + + P T	+ + + P T	+ + + P T
1098	+ + + + +	+ P − − −	+ P P T −	+ P P T −	T T − − −
1099	+ + + + +	+ + P − −	P P P P T	P P P T T	+ + P T −
1100	+ + + + +	+ + + P −	+ + + + +	+ + + + +	+ + + + +
1025	+ + + + +	+ + P T −	+ + + + +	+ + + + +	+ + + P T

*Date of slaughter.

Discussion of Results

The foregoing data represent the results of a study of 345 head of hogs showing agglutinins in their blood for Brucella and situated in three widely separated herds.

By maintaining and observing the herds under different systems of management, considerable information was obtained as to the course, spread and control of the disease. The data strongly indicate that Brucella infection in swine is of a self-limiting nature, and should be controlled or eradicated easily from a herd within a short period of time. The practice of adding young animals to a group of hogs in which infected animals are present appears to be the important factor in maintaining the disease in the herd indefinitely.

The agglutination test, as has already been pointed out by others, is a satisfactory means of detecting Brucella infection in a herd. One cannot, however, be guided by any given agglutination titre based on one test in establishing a line between the infected and non-infected. In a herd containing infected animals, a partial agglutination in a 1:25 dilution may have just as much significance as a complete reaction in a 1:500 dilution. Further, the cultural findings and high agglutination titres cannot be correlated. One may readily obtain the organism in many different organs in one animal reacting in a high titre and obtain negative cultural findings in another hog showing the same titre.

The cultural findings on 46 hogs are conclusive in showing that the organism is distributed widely throughout the body of the infected hog.

In view of the fact that more than two years of observation in these three herds, as well as in 12 others in the state of Michigan, has revealed abortions due to Brucella to be of no consequence, the question that may be raised is: "What is the importance of detecting Brucella infection in hogs and the necessity of eradicating it from a herd?" In so far as the hog is concerned, the time may arrive when the disease will result in an increasing number of abortions and considerable arthritis. It is a matter of fact that in certain sections of this country the occurrence of abortions in infected pregnant females is already a serious problem. It appears to us that, at the present time, Brucella infection in swine is as much if not a more important problem from the standpoint of human health as it is an economic problem in the raising of swine.

There has been plenty of evidence produced to show that *Br. suis*, of which the hog is the chief host, is as pathogenic for man as is *Br. abortus* or *Br. melitensis*, if not more so. A large number of the cases of undulant fever that have occurred in man in this country have been due to *Br. suis*. While many of the cases due to this species have a history of having been in contact with hogs, on the other hand, many of them have not. The possibility of the disease being contracted through the handling or ingestion of hog meat is a question which we have studied for some time. The results of these studies will be presented at an early date.

SUMMARY

There have been presented data on the nature of natural Brucella infection in 345 head of hogs showing agglutinins in their blood for Brucella.

The hogs were situated in three separate herds maintained under different conditions from the standpoint of controlling the disease.

The data indicate that Brucella infection of the hog is a self-limiting disease and may be brought under control or eliminated from a herd by means of the blood-test made at intervals of 30 days. All reactors, even those reacting to a slight degree, should be separated from the negative reacting ones.

The causative organism is well distributed throughout the body of the infected hog.

The only species of Brucella found occurring in naturally infected hogs was *Brucella suis* (Traum).

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A TISSUE VACCINE FOR HOG CHOLERA*

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INTRODUCTION

The new tissue vaccine for hog cholera which is to be described in this paper is the culmination of experiments and observations on that disease at the University of California Agricultural Experiment Station, from 1925 to 1932.

EARLY EXPERIMENTS

Since marked success followed the use of a vaccine developed by the author[1,2] for rinderpest, it was believed possible that this method, or an adaptation of it, might be applicable to hog cholera. Accordingly, in the first experiments a vaccine was prepared employing the same tissue as for rinderpest, namely, spleen, lymph-glands, heart, blood and liver chiefly, and the same attenuating agents, namely, phenol, or phenol with heat, and agitation in a shaking-machine for from 12 to 48 hours.

The numerous hog-cholera vaccines prepared by these modifications of the method for rinderpest were tested on highly susceptible pigs. Some of the vaccines were found to be insufficiently attenuated. After storage in the refrigerator for a period of from 3 to 4 months, they lost potency and failed to protect pigs against exposure to virus. A few experiments gave promising results, but the potency of the vaccine was too transient to be relied upon.

A modification, developed by Kelser,[3] namely, preparing rinderpest vaccine by attenuation with chloroform, is claimed to be superior to the vaccine with phenol as the attenuating agent. This modification was applied to the preparation of hog-cholera vaccine, but was discontinued after several trials which resulted unfavorably.

Kakizaki and his associates,[4] who have been working on rinderpest vaccines in Korea for a considerable number of years, report good results with the use of toluol as the attenuating agent. Jacotot,[5] in Indo-China, confirms their findings. In several experiments performed in this laboratory, a few encouraging results were obtained with toluol, but they were not consistently dependable.

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Formalin, used by Laidlaw and Dunkin,[6] in the preparation of dog-distemper vaccine, was tried in several experiments, but no favorable results were obtained. A report from the Chief of the Bureau of Animal Industry[7] reveals that investigators from the Bureau encountered the same difficulty with the use of formalin in the preparation of a vaccine for hog cholera. They followed the method of Laidlaw and Dunkin as nearly as possible. Benner[8] reports similar results.

It appears from the observations made on the modifications tried in this laboratory that none of the above-mentioned attenuating agents in combination with the tissues used for vaccines is particularly applicable to hog-cholera virus.

During 1929, a fifth attenuating agent was tested. This was eucalyptus oil, suggested by Kakizaki and his associates,[4] who used it for rinderpest vaccine in one experiment. Although they claimed encouraging results, they did not, at that time, carry the work further. About this time, some changes were made in the tissues employed by the writer. Red marrow of bone was added and, occasionally, portions of lung showing petechiae, kidneys, thymus and pancreas. The first really successful experiments at the University of California followed the trial of vaccines containing red marrow in the tissue pulp and eucalyptus oil as the chemical attenuating agent. In all subsequent experiments on pigs protected with this type of vaccine and then exposed to hog-cholera virus under laboratory conditions, not a single mortality has occurred in 51 swine. Two important contributing factors to a satisfactory vaccine appear, then, to be red marrow of bone and eucalyptus oil.

Preparation of the Vaccine

Type of pig used and method of handling: A healthy, highly-susceptible pig, weighing approximately 40 pounds, is the best type of animal to use for the preparation of hog-cholera vaccine. In a pig of this size and age, the bones are not so ossified as in larger and older animals, and the red marrow of bone may be obtained more easily.

This type of animal is injected with hog-cholera virus. Close observation is made upon it for symptoms and its temperature is recorded at morning and evening.

The animal should show a rise in temperature within a few days, and, on the third or fourth day after the initial rise, the pig should develop such pronounced symptoms of hog cholera as inappetence, drowsiness, muscular tremors and, possibly, fits

and vomiting. When two or more of these symptoms are observed, the animal is killed and all the lymph-glands, the spleen and the long bones containing a large amount of red marrow, are removed as aseptically as possible. Occasionally, the heart, kidneys, thymus, pancreas, tonsils, portions of liver and of lungs which present marked petechiae are added.

Preparation of tissue for vaccine: All of the fat, the heavy connective tissue, and fascia are removed from these organs and the red marrow from the bones. These tissues are placed in a vessel, containing a 5 per cent solution of phenol, for 10 minutes, with occasional stirring to insure disinfection of all surfaces which may have become contaminated with microörganisms during the removal of the organs or during subsequent handling of them. The tissues are then rinsed in sterile tap water to remove the excess of phenol. (All of the utensils are sterilized in the autoclave or thoroughly soaked in disinfectant.) The tissues are cut into small pieces and first passed through a food-chopper, then through a Quaker City corn-mill, which cuts and macerates the tissue into smaller particles and also removes most of the calcified material from the red marrow. Next, this ground mixture is passed through a Matthews or Latapie tissue-mill, which reduces it to a still finer consistency. The tissue should be passed through the mill two or three times to complete maceration. This mixture is then forced through a strainer of approximately 1/12-inch mesh by means of a pestle. In this manner, the parenchymatous tissue is separated from the connective tissue which is left in the strainer and discarded.

Preparation of vaccine from macerated and ground tissue: Several types of vaccine have been prepared by varying the diluting agent in which the tissue is suspended. The most potent, in both protective and keeping qualities, is prepared by taking three parts, by weight, of tissue and adding one part, by volume, of pure glycerin (pH 7.4). Practically the same results are obtained by taking one part, by weight, of tissue and adding an equal part, by volume, of a solution composed of equal parts of glycerin (pH 7.4) and 0.85 per cent sodium chlorid solution (pH 7.4). The latter suspension, more liquid than the glycerin, facilitates mixing and administering the vaccine.

In an endeavor to obtain a less expensive suspension fluid, a gelatin-saline solution (pH 7.4), composed of 0.85 per cent sodium chlorid and enough gelatin to give the liquid a viscosity sufficient to hold the tissue in suspension, was tried on numerous occasions. In every instance the vaccine lost in potency and keeping qualities.

Method of bottling and attenuating the vaccine: A certain amount of the prepared tissue mixture is measured into a graduated container. This material is transferred to sterile bottles, and the desired amount of eucalyptus oil is added to the contents of each bottle. The bottles are stoppered tightly and shaken for several minutes to procure thorough commingling of the attenuating agent and the tissue mixture. These bottles then are stored in the refrigerator for attenuation, which is sufficient usually after a period of from 1½ to 2 months, depending upon the percentage of eucalyptus oil present; the lower the percentage of this agent, the longer the time required. It is advisable to shake well each bottle of vaccine for a few moments every day during the first 8 or 10 days of the attenuation period.

Mixing vaccine prior to potency test: Before testing the vaccine on pigs, each bottle of a given lot should be emptied into a large sterile container and mixed thoroughly for several minutes, so that each cubic centimeter of vaccine may have the same potency. Fresh sterile bottles are filled with this mixed vaccine, corked tightly, and stored in the refrigerator, ready for use.

Test for potency: Each lot of vaccine should be tested on highly susceptible pigs. It is advisable to test the virulence of the vaccine on two or three pigs before vaccinating several animals, to insure sufficient attenuation.

In testing the potency of each vaccine, the use of five pigs is recommended and two injections, if vaccination is by the intramuscular route, or one injection if vaccination is by the intraperitoneal route. One animal should be kept as a control and left in contact with the four others during the period in which they are developing resistance, to prove that no transmission of hog cholera occurs. When the intramuscular method is used, the two vaccinations may be given one or two weeks apart.

Following vaccination, at least two weeks should elapse before the virus is injected into the protected pigs and the control, since it has been observed from early trials that pigs develop resistance against hog cholera rather slowly. If a sufficiently large dose of potent vaccine has been given pigs, they will exhibit little or no effect from the injection of hog-cholera virus, but the control pig will develop the disease promptly.

Method of Vaccination

Intramuscular route: The most favorable area for vaccination is the fleshy part of the neck, just in front of the shoulder-

blade. The area is disinfected and the needle of the syringe inserted an inch or more into the muscular tissue before the vaccine is injected. Immediately following the injection, this area is massaged thoroughly to spread the vaccine through the muscular tissue and produce a more rapid absorption. The second injection is administered on the opposite side of the neck.

Intraperitoneal route: The pigs should not be fed for several hours previous to vaccination, so that the stomach and intestines may become partially emptied. The pig is suspended by its hind legs. In this position its stomach and intestines will gravitate toward the diaphragm; massage of the abdomen downward will facilitate this. An area is disinfected midway between the inner side of the thigh and the median line of the abdomen. The needle is inserted through the abdominal muscles into the peritoneal cavity and the vaccine injected. It is advisable to warm the vaccine to body temperature before injection. If the vaccine is too cold, the animal is likely to suffer from shock.

Dosage of Vaccine

The dosage depends on the type of vaccine used and the size of the pig to be protected. From 5 to 10 cc of the less concentrated form, which contains 0.5 gm of tissue per cc, is usually sufficient to protect an animal weighing 30 or 40 pounds. For a 100-pound or heavier pig, a dose of from 10 to 15 cc is suggested. The concentrated form, which contains 0.75 gm of tissue per cc, must be diluted with the glycerin-saline diluting fluid to a dose equivalent to that of the less concentrated form.

Experimental Application of Hog-Cholera Vaccine

The following experiments show the results of tests on pigs inoculated with various lots of vaccines. Some of the vaccines contain fewer organ tissues than others, and the content of the attenuating agent varies. Other differences in the experiments are in the age of the vaccine, the dosage, or the number and route of injections.

Experiment 1

Vaccine lot 1: This vaccine was prepared on May 10, 1929. Tissues used: spleen, lymph-glands, tonsils, red marrow of bone, heart, and portions of lung showing petechiae. The tissue pulp was divided into two portions:

Lot I(a): Volume of tissue pulp plus a small amount of	
Glycerin	400 cc
Eucalyptus oil (1%)	4 cc

Lot I(b): Volume of tissue pulp plus a small amount of
Glycerin ..... 200 cc
Eucalyptus oil (1.5%)..... 3 cc

The results with this lot of vaccine are shown in table I.

In this experiment a vaccine was produced which was found potent when tested on twelve pigs, in three series, at intervals

TABLE I—*Results of tests of vaccine 1, lots I (a) and I (b).*

PIG	VACCINATION				TEST FOR SUSCEPTIBILITY TO VIRUS	RESULTS
	DATE OF INJECTION* (1930)	VACCINE				
		AGE (DAYS)	LOT	DOSE (CC)		
73	1st: Jan. 3	238	I(b)	5.0	Jan. 27	73 and 74: slight temperature reaction after virus injection; rapid return to normal
74	2nd: Jan. 13	248	I(b)	7.5		
	No control					
75†	1st: Feb. 11	277	I(b)	5.0		75 and 76: no reaction
76						77, 78 and 79: slight temperature; reduced appetite on 7th and 8th days after virus injection, followed by speedy return to normal
77						
78	2nd: Feb. 25	291	I(a)	5.0	Mar. 11	
79						
80	Control	—	—	—		80 (control): temperature on 4th day; severe case of hog cholera; died on 9th day
82†						82 to 86: no reaction in any of the vaccinated pigs
83	1st: Apr. 21	346	I(b)	4.0		
84						81 (control): temperature on 3rd day; developed fits on 7th day and was killed
85	2nd: May 5	360	I(b)	4.0	May 20	
86						
81	Control	—	—	—		
87†						Severe temperature reactions in all of the vaccinated pigs. 87, 88 and 89 received 50 cc of anti-hog cholera serum. 90 and 91 were left untreated. Four days after the rise in temperature, all the pigs were back to normal; untreated recovered as quickly as treated
88						
89	June 3	389	I(b)	6.0	June 24	
90						
91						
92	Control	—	—	—		92 (control): temperature on 3rd day; severe case; developed fits on 7th day and was killed

*All injections made intramuscularly.
†Pigs kept in same pen throughout the experiment.

of approximately eight months to a year. It will be noted that the reactions to virus are very mild, consisting usually of a slight rise of temperature. However, in pigs 77, 78 and 79, a reduced appetite as well was observed. An attempt to produce resistance with a single intramuscular injection by raising the dosage slightly and allowing three weeks instead of two to elapse before inoculating virus was not very successful, although the pigs which received anti-hog cholera serum as a therapeutic measure recovered no more promptly than the two which were left untreated.

EXPERIMENT 2

Vaccine lot 2: This vaccine was prepared on October 16, 1930. Tissues used: spleen, lymph-glands and red marrow of bone.

Tissue pulp	280 gm
Glycerin (pH 7.6)	90 cc
Volume of mixture	360 cc
Eucalyptus oil (5%)	18 cc

The results of tests on lot 2 appear in table II.

TABLE II—*Results of tests of vaccine 2.*

PIG	VACCINATION			TEST FOR SUSCEPTIBILITY TO VIRUS	RESULTS
	DATE OF INJECTION* (1931)	VACCINE			
		AGE (DAYS)	DOSE (CC)		
97	1st: Jan. 14 2nd: Jan. 21	90 97	5.0 9.0	Feb. 5	No reaction to virus injection
98†					98: slight temperature on 5th and 6th days after virus injection; normal on seventh day
99	1st: Feb. 16	123	8.0		
100					
101	2nd: Feb. 24	131	8.0	Mar. 8	99: slight temperature on 7th and 8th days; normal on 9th day
102	Control	—	—		100 and 101: no reaction 102 (control): high temperature on 4th day; severe case; killed on 8th day

*All injections made intramuscularly.
†Pigs kept in same pen throughout the experiment.

A vaccine containing 5 per cent of eucalyptus oil was potent when tested on five pigs in two series at intervals of three to four months. The control pig for the second series served for the first also, since the same virus was used. The results on the

pigs in this experiment were about the same as in the preceding experiment.

EXPERIMENT 3

Vaccine lot 3: This vaccine was prepared on April 15, 1931. Tissues used: spleen, lymph-glands, tonsils, pancreas, heart, kidneys, red marrow of bone, a piece of liver, and portions of lung showing petechiae.

Tissue pulp and diluting fluid	1,265 gm
Glycerin (pH 7.6)	425 cc
Volume of mixture	1,550 cc
Eucalyptus oil (4%)	62 cc

The effect of this lot of vaccine is shown in table III.

TABLE III—*Results of tests of vaccine 3.*

PIG	VACCINATION: DATE OF INJECTION (1931)	VACCINATION: VACCINE AGE (DAYS)	VACCINATION: VACCINE DOSE (CC)	TEST FOR SUSCEPTIBILITY TO VIRUS	RESULTS
104	1st*: July 14	90	10.0	Aug. 13	104: slight temperature on 6th day after virus injection; return to normal on following day
105	2nd*: July 29	105	10.0		105: no reaction
106	Control	—	—	Sept. 10	106 (control): high temperature; severe case; developed fits on 6th day and was killed
107	Control	—	—		107 (control): high temperature; severe case; killed on 6th day
†	1st*: Aug. 22	129	5.0	Sept. 14	Seven pigs: no visible symptoms, although no temperature was taken
	2nd*: Aug. 29	136	5.0		
108	Nov. 12‡	211	10.0	Dec. 2	108: slight temperature on 6th day after virus injection; quick return to normal
109					109: no reaction
110	Control	—	—		110 (control): high temperature; severe case; developed fits and was killed on 8th day

*Injections made intramuscularly.
†Seven pigs used in this test and kept under field conditions. Pigs 106 and 107 were used as controls for this experiment also.
‡Injection made intraperitoneally. Both pigs received a severe shock from this injection of cold vaccine but appeared normal on the following day.

Eleven pigs were tested with this lot of vaccine containing 4 per cent of eucalyptus oil. The first series of two pigs received vaccine preserved for three months; the second series of seven

animals was given a 4-month-old vaccine. This series was kept under field conditions; their temperatures were not recorded. The third series of two pigs received an intraperitoneal injection of this vaccine preserved for 7 months. The results of this experiment show that a vaccine was prepared which was potent at 7 months.

EXPERIMENT 4

Vaccine lot 4: This vaccine was prepared on September 17, 1931. Tissues used: spleen, lymph-glands, red marrow, heart, kidneys, a small piece of liver, and a portion of lung showing petechiae.

Tissue pulp	1,755 gm
Glycerin	585 cc
Glycerin diluting fluid	1,000 cc
Eucalyptus oil (3.25%)	102 cc

The results of the test on this lot of vaccine are shown in table IV.

TABLE IV—*Results of tests of vaccine 4.*

PIG	VACCINATION			TEST FOR SUSCEPTIBILITY TO VIRUS	RESULTS
	DATE OF INJECTION*	VACCINE			
		AGE (DAYS)	DOSE (CC)		
111	1st: Dec. 29, 1931	103	7.0	Jan. 20	111 and 112: no reaction to virus
112	2nd: Jan. 6, 1932	111	10.0		

*Injections made intramuscularly.

In this experiment, vaccine containing 3.25 per cent of eucalyptus oil was potent when tested at 3½ months. There was no control pig but the virus used was perfectly fresh. The results on the pigs were practically the same as in the three preceding experiments.

EXPERIMENT 5

Vaccine lot 5: This vaccine was prepared on March 5, 1932. Tissues used: spleen, lymph-glands, red marrow, and a small piece of liver.

Tissue pulp	500 gm
Gelatin-diluting fluid (pH 7.4)	500 cc
Volume of mixture	975 cc
Eucalyptus oil (3%)	29.25 cc

The effect of this vaccine on pigs appears in table V.

TABLE V—*Results of tests of vaccine 5.*

PIG	VACCINATION			TEST FOR SUSCEPTIBILITY TO VIRUS	RESULTS
	DATE OF INJECTION* (1932)	VACCINE AGE (DAYS)	VACCINE DOSE (CC)		
119	1st: Apr. 18	44	10.0	May 10	119: temperature on 5th day after virus injection; prompt return to normal
120	2nd: Apr. 26	52	10.0		120: temperature on 5th day; reduced appetite on 5th and 6th day, followed by return to normal
121	Control	—	—		121 (control): high temperature; severe case; killed on 7th day

*Injections made intramuscularly.

This vaccine, containing a 3 per cent solution of eucalyptus oil, was tested at 1½ months on a series of two pigs. One of the pigs reacted a little more severely to the virus than most of the animals tested, except pigs 77, 78 and 79, of the first experiment previously referred to. This seems to indicate a slight reduction in potency, traceable probably to the vaccine content.

EXPERIMENT 6

Vaccine lot 6: This vaccine was prepared on April 13, 1932. Tissues used: spleen, lymph-glands, red marrow, heart, kidneys and a small piece of liver.

Tissue pulp	850 gm
Gelatin-diluting fluid (pH 7.4)	850 cc
Volume of mixture	1,650 cc
Eucalyptus oil (2%)	33 cc

The results of tests on lot 6 are found in table VI.

Vaccine, containing 2 per cent of eucalyptus oil, was tested at 1½ months. Both pigs showed a little rise in temperature. The fact that both had a mild reaction may indicate a less potent vaccine.

EXPERIMENT 7

Vaccine lot 7: This vaccine was prepared on May 17, 1932. Tissues used: spleen, lymph-glands, red marrow, and a small piece of liver.

Tissue pulp	875 gm
Gelatin-diluting fluid (pH 7.4)	875 cc
Volume of mixture	1,725 cc
Eucalyptus oil (2%)	34.5 cc

The results of tests on lot 7 may be seen in table VII.

TABLE VI—*Results of tests of vaccine 6.*

PIG	VACCINATION			TEST FOR SUSCEPTIBILITY TO VIRUS	RESULTS
	DATE OF INJECTION* (1932)	VACCINE			
		AGE (DAYS)	DOSE (CC)		
122	1st: May 31	48	7.0		122: slight temperature on 5th and 6th days after virus injection; rapid return to normal
123	2nd: June 8	56	10.0	Jan. 28	123: slight temperature on 6th day, followed by return to normal
126	Control	—	—		126 (control): high temperature; severe case; killed on 7th day

*Injections made intramuscularly.

TABLE VII—*Results of tests of vaccine 7.**

PIG	VACCINATION			TEST FOR SUSCEPTIBILITY TO VIRUS	RESULTS
	DATE OF INJECTION† (1932)	VACCINE			
		AGE (DAYS)	DOSE (CC)		
127				Sept. 7 (44 days)	127 and 128: temperature on 5th day after virus injection; normal on 7th day
128					
137‡					137 (control); high temperature; severe case; killed on 8th day
129				Oct. 27 (94 days)	129: temperature on 5th day after injection; prompt return to normal
130	1st: July 11	55	15.0		130: very slight temperature on 5th day; rapid return to normal
138‡					138 (control): high temperature; severe case; killed on 7th day
131				Dec. 6 (134 days)	131 and 132: high temperature for 3 days after virus injection; reduced appetite; speedy return to normal
132					
143‡	2nd: July 25	69	15.0		143 (control): high temperature; severe case; killed on 8th day.
133				Apr. 24, 1933 (273 days)	133: high temperature on 4th day after injection; rapid return to normal
134					134: slight temperature on 6th day after injection; prompt return to normal
135					
136§					

*This experiment is in progress.
†Injections made intramuscularly.
‡Control.
§Pigs 127 to 136 kept under field conditions.

The purpose of this experiment, which is now in progress, is to determine the length of the resistance produced. Hog cholera virus was inoculated into two of the vaccinated pigs, 44 days after the second vaccination; into two others, 94 days after the second vaccination; into two others, 134 days after the second vaccination, and into two others, 273 days after the second vaccination. Each of these pigs exhibited a rise in temperature and the two given virus 134 days following their second vaccination lost appetite as well, but all quickly returned to normal. This tends to prove that pigs are resistant for at least nine months. The length of resistance will be more definitely established in a later report.

EXPERIMENT 8

Vaccine lot 8: This vaccine was prepared on July 7, 1932. Tissues used: spleen, lymph-glands, heart, kidneys, and a small piece of liver.

Tissue pulp	50 parts by weight
Gelatin-diluting fluid (pH 7.4)	50 parts by volume
Eycalyptus oil	1.5% of volume of mixture

The effect of this vaccine on pigs is shown in table VIII.

TABLE VIII—*Results of tests of vaccine 8.*

PIG	VACCINATION			TEST FOR SUSCEPTIBILITY TO VIRUS	RESULTS
	DATE OF INJECTION (1932)	VACCINE			
		AGE (DAYS)	DOSE (CC)		
139	1st*: Nov. 8	124	15.0	Dec. 6	139: slight temperature on 5th day after virus injection; returned to normal
140	2nd*: Nov. 15	131	15.0		140: temperature on 3rd day; lost appetite; died on 12th day
143†	Control	—	—		143 (control); high temperature; severe case; killed on 8th day
141					141 and 142: temperature on 3rd day after virus injection; appetite reduced for two days; quick return to normal
142	Nov. 14‡	130	15.0	Dec. 6	
143†	Control	—	—		

*Injections made intramuscularly.
†This pig was used as a control also for pigs 131 and 132 in tests of vaccine 7 (see table VII).
‡Injection made intraperitoneally.

In this experiment it will be seen that one of the two pigs vaccinated intramuscularly died as a result of the injection of

virus. The two pigs vaccinated intraperitoneally reacted more strongly to virus than the two animals vaccinated by the same route in experiment 3. This vaccine contained no red marrow of bone.

Field Trial at Davis

On October 5, 1931, 16 pigs at the University Farm, Davis, were vaccinated with lot 3, which had been tested on nine pigs at the laboratory and proved to be potent. (See table III, pigs 104 and 105, and seven pigs kept under field conditions at Berkeley.)

Five of the pigs at Davis were being used in a mineral-deficiency experiment, were unthrifty in appearance and some were scouring. The remaining 11 pigs were the offspring of abortus-reacting sows and were housed with their mothers in a shed in which a number of porcine and bovine reactors were quarantined. All of the sows and other pigs in this shed, except the 11 offspring and two controls, were immunized against hog cholera by the serum-virus method when they were suckling pigs.

Each of the 16 pigs received 5 cc of the vaccine, intramuscularly. On October 12, 1931, eleven of the pigs received a second vaccination.

On November 4, 1931, the 16 pigs (of which 11 had received two vaccinations and 5 only one vaccination) and two controls received 1 cc of virus, intramuscularly. All developed a severe reaction to the hog cholera virus. The two controls and eight of those vaccinated died. All of the previously immunized pigs in contact with the vaccinated ones became sick and 40 per cent of them died of hog cholera.

When these results were observed, two pigs at Berkeley were given vaccine taken from the same bottle that had been used at Davis. These two animals (pigs 108 and 109, table III) received one vaccination each, intraperitoneally, and later were proved to be highly resistant to hog cholera.

The question arises: Can pigs be satisfactorily protected against hog cholera when they are unthrifty or when they are kept under the conditions described?

Discussion

From these experiments, it is evident that a definite resistance was produced against hog cholera. The efficacy of this vaccine appears to depend on the presence of two essential ingredients: from 1.5 to 5 per cent of eucalyptus oil as the attenuating agent

and formative tissue, particularly red marrow of bone, as well as the lymphatics. The fact that, in experiment 8, the two pigs injected intraperitoneally with a vaccine which contained no red marrow and was not suspended in glycerin exhibited a more severe reaction to virus than those vaccinated in the same manner in experiment 3 and that the only mortality recorded occurred in this experiment in an intramuscularly vaccinated animal stresses the importance of this tissue and the preserving fluid. It is the firm opinion of the author that the most potent vaccines are prepared from lymph-glands, red marrow of bone, spleen, and, to a less extent, perhaps, pieces of lung showing marked petechiae when they are suspended in glycerin or a glycerin-saline solution. The presence of other tissues tends to reduce the potency somewhat. Also, the gelatin-saline diluting fluid appears to be inferior to the glycerin one in this respect. The results of experiments 5, 6 and 8 tend to indicate this, and a study of the temperature charts of all the vaccinated pigs leads to this conclusion, although it cannot be shown very clearly in this paper.

Proper administration of a single dose of vaccine intraperitoneally seems as effective as two intramuscular doses. In experiment 1, it will be seen that a very severe reaction was produced when virus was injected into pigs which had received but one vaccination by the intramuscular route. Although the two pigs which were untreated recovered as quickly as those which were given anti-hog cholera serum, this single intramuscular vaccination failed to give adequate protection against virus.

The vaccine described has no therapeutic effect on a sick pig or on one in the incubation period of hog cholera. It is purely a prophylactic measure with the advantage of being cheaply prepared, of retaining its potency for a considerable length of time and, particularly, of producing a very definite resistance in a safe manner. The danger of transmitting hog cholera to highly susceptible pigs, which frequently occurs in the serum-virus method, is eliminated. It has been proved in experiments 1 and 2 that vaccine prepared by the method detailed in this paper did not transmit the disease to susceptible animals through cohabitation with those that were undergoing vaccination.

SUMMARY

Early experiments with vaccines prepared from the same tissues used by the author for rinderpest vaccine and using, as attenuating agents, phenol or phenol with heat and agitation by

shaking; chloroform, toluol, and formalin failed to yield satisfactory results.

Red marrow of bone was added to the vaccine and eucalyptus oil was used as the attenuating agent. Vaccine prepared in this manner gave successful results when tested on pigs under controlled conditions.

The preparation of this vaccine is given in detail.

The use of five pigs for testing the potency of each vaccine is recommended and two intramuscular injections, one or two weeks apart, or one intraperitoneal injection. At least two weeks should elapse before virus is inoculated, since it has been observed that pigs develop resistance to hog cholera rather slowly.

The two methods of vaccination used, intramuscular and intraperitoneal, are described.

The dosage of vaccine varies according to the content of tissue by weight and the body weight of the pig.

In eight experiments described, resistance tests were made on pigs to determine the potency of eight lots of vaccine. Every vaccine except lot 8 was decidedly potent. The potency of lot 8, which lacked red marrow and glycerin, was considerably lessened. In experiments 5 and 6, a slightly reduced potency was observed. Experiment 7, still in progress, attempts to establish the length of the resistance produced.

A field trial at Davis, on five pigs used in a mineral-deficiency experiment, and 11 offspring of Bang-disease reactors, resulted in a 50 per cent mortality, although the vaccine was tested before and after the trial and found to be potent. In spite of the fact that they had been immunized previously by the serum-virus method, all of the pigs in contact with the vaccinated animals became ill and 40 per cent died.

Results of these experiments indicate that a definite resistance was produced and that essential factors for a potent vaccine are: from 1.5 to 5 per cent of eucalyptus oil, as the attenuating agent; and formative tissue, particularly red marrow of bone, as well as the lymphatics. The presence of other tissues appears to lessen the potency of the vaccine somewhat; gelatin-saline diluting fluid may be inferior to glycerin-saline diluting fluid in this respect.

One intraperitoneal injection, if properly administered, appears as effective as two intramuscularly.

The vaccine described has no therapeutic effect on a sick pig or on one in the incubation period of hog cholera. It is simply

a prophylactic measure. No transmission of hog cholera occurred through cohabitation of susceptible pigs with animals which were undergoing vaccination.

ACKNOWLEDGMENTS

The writer wishes to express his cordial thanks to Dr. Frederick W. Wood, of Cutter Laboratory, Berkeley, for his kindly aid in furnishing housing quarters and other facilities over a period of years for pigs used in numerous preliminary experiments; to Miss Gladys M. Woods, of the Division of Veterinary Science, for assistance in preparing vaccines and recording results; and to Mr. Thomas L. Bean and Mr. Herman F. Hofliger, also of the Division, for their painstaking care in handling the animals under experiment.

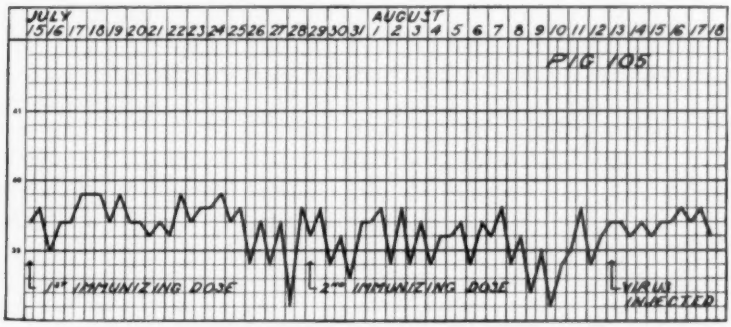

FIG. 1. Temperatures of pig 105. Previous to injection of virus, this animal received two intramuscular injections of hog cholera vaccine which was 90 and 106 days old, respectively.

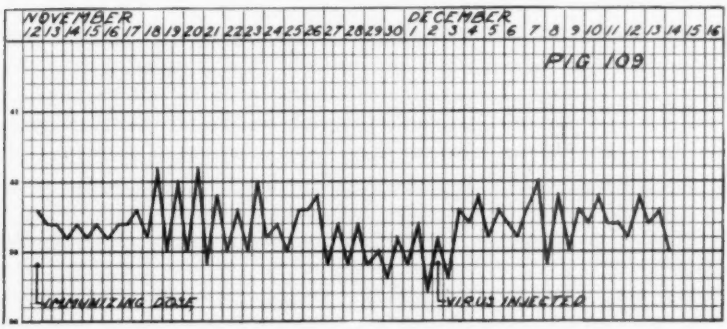

FIG. 2. Temperatures of pig 109. Before receiving an injection of virus, this animal was immunized intraperitoneally with one dose of hog cholera vaccine which was 211 days old.

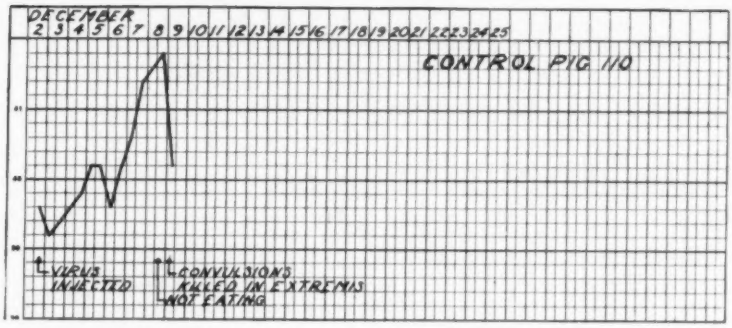
DECEMBER
2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25
CONTROL PIG 110
VIRUS INJECTED
CONVULSIONS
KILLED IN EXTREMIS
NOT EATING

FIG. 3. Temperatures of control pig 110, following injection of virus. This chart is typical of the charts of all the control animals used in the experiments cited.

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DISCUSSION

DR. C. N. MCBRYDE: I listened to Dr. Boynton's paper with a great deal of interest, because we, in the Bureau, have also been interested in tissue vaccines. We started this work not because we believed that the simultaneous immunization of pigs with serum and virus tends to spread hog cholera, but rather because we had in mind the possibility of developing a cheaper method of immunization.

We started experiments along this line some years ago at the Field Station of the Biochemic Division of the U. S. Bureau of Animal Industry at Ames, Iowa, which is under the supervision of Dr. Marion Dorset, at Washington, D. C. We have done a considerable amount of work with tissue vaccines, extending from 1927 to 1930. Vaccines were prepared from the tissues of cholera-infected pigs, using varying percentages of different attenuating agents. We used spleen, lymphatic glands, liver and kidneys. Our results, briefly, seemed to indicate the possibility of obtaining protection at times by any one of several different methods, but the results were irregular and not dependable, and we have never felt that we could recommend the use of such vaccines in practice. We believe that a fair conclusion at this time would be that thus far no vaccine has been shown to be of sufficient reliability to warrant its employment in practice.

DEVELOPMENTS CONCERNING HOG CHOLERA*

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In order that there may be no confusion as to what we are discussing when we speak of hog cholera, it may be well to devote a few moments to the mental picture which comes to our mind when hog cholera is mentioned. The word "cholera" means, literally, "from bile," and was applied to the disease when it was regarded primarily as an affection of the intestinal tract. With the development of knowledge and technic in the field of bacteriology, the intestinal tract naturally became the center of study, and a pathogenic organism, *Salmonella suipestifer (B. suipestifer)*, was isolated and after considerable experimentation was designated as the etiological agent responsible for the disease. In some outbreaks, especially during certain seasons of the year, pneumonia was quite common, and there were found in these diseased lungs bipolar organisms differing in many essential features from *S. suipestifer*. Those cases where pneumonia played a prominent part were designated as swine plague. We had, therefore, in the light of these early experiments, two highly contagious diseases of swine, *viz.*, hog cholera, an affection of the intestinal tract, and swine plague, a disease of the respiratory system.

Doubt as to the correctness of the above conception was raised, because of the unsatisfactory results of immunization experiments in swine as compared with laboratory animals. No doubt the filtrable virus was an unknown factor in most of the outbreaks of both hog cholera and swine plague, investigated in the earlier years. It was the discovery of the filtrable agent, hog cholera virus, which forced us to revise all our previous conceptions of swine diseases, especially hog cholera and swine plague, and likewise has made our present nomenclature inappropriate.

Since the incidence of swine erysipelas, especially in the Corn Belt, is increasing, it has become more vital that we should be able to define and differentiate the various swine diseases. In typical cases, as a rule, this is not difficult. The deviation from the classical, however, is what is liable to lead to confusion, error and consequent loss.

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*Presented at the seventieth annual meeting of the American Veterinary Medical Association, Chicago, Ill., August 14-18, 1933.

For many years, hog cholera has been defined to our students as follows, *viz*: A septicemia, usually acute, characterized by petechial hemorrhages in the skin, lymph-glands and internal organs of the body. The cause is a filtrable virus which cannot be transmitted to other than porcine species. Being primarily a septicemia, there is no pectoral or intestinal form of hog cholera. An enteritis or pneumonia, if one or both be present, is merely a complication of the septicemia caused by the filtrable virus. They are secondary and due to the presence of other infectious agents than the hog cholera virus. Enteritis, particularly, has a high incidence entirely independent of hog cholera.

The work which has been done during the past few years verifies our earlier conception of hog cholera, *i.e.*, that it is primarily a septicemia characterized by hemorrhages, usually petechial, resulting from injury to the vessel walls. Emphasis on the secondary changes has led to much confusion and not a little loss.

Hog cholera has been referred to as a lesionless disease. This is as erroneous as calling a secondary enteritis the intestinal form of hog cholera.

Action of the Virus

Hog cholera virus, like some other filtrable viruses, notably that of spotted fever of man, attacks first the endothelium of the arterioles and capillaries. Only after these have been injured, can we find changes in the organs themselves. Since the primary action of the virus is the production of a degeneration in the walls of the arterioles and capillaries, petechial hemorrhages have an important diagnostic significance and should never be disregarded. In the nervous system, as in other organs of the body, the principal lesion is injury to very small blood-vessels in the central nervous system, which leads to the nervous symptoms so common in this disease. These do not appear as a general nervous disturbance, but as more or less typical symptoms due to lesions in certain areas of the central nervous system. These lesions have been found to be a non-suppurative meningoencephalomyelitis, in which both the white and the gray matter are involved and are found in about 75 per cent of the cases. There seems to be no preferred location for the lesions in the brain, as they may be found in all parts. In addition to the injury to the vessel wall, there is a perivascular infiltration. Small arterioles sometimes show changes principally at bifurcations, as early as the second day following inoculation. Small veins are less frequently affected. These changes may be accompanied by an infiltration of the pia and choroid plexus. By

some, the lumbar cord is considered the part of the central nervous system most inclined to be involved. Whether this is the explanation for the incoördination of the rear limbs so commonly seen in hog cholera is as yet not decided.

Chronic cases show the same fundamental changes as the acute, but the lesions are less extensive.

Quick Method of Diagnosis Sought

A rapid but accurate method of determining whether or not hog cholera virus is present, in many cases where history and lesions are not decisive, has been the hope of many for a long time. Much valuable work has been done during recent years on the histopathology of hog cholera by Bel,[1] Cain and Seifried,[2] David and Schwarz,[3] Nieberle,[4] Rohrer,[5,6] and others. Lesions, in order to be of value, diagnostically, must be specific, or at least sufficiently characteristic to differentiate the disease from those which tend to confuse us in making a differential diagnosis. In a number of diseases caused by filtrable viruses, we are able to assist very materially in identifying the disease by characteristic cellular inclusions which are not present in normal tissue. Attempts have been made to find some characteristic cellular inclusions, or at least some sufficiently characteristic cellular changes, to enable us to have a useful rapid method of diagnosis in hog cholera. There are cellular inclusions in Borna disease and dog distemper, along with the perivascular infiltration in the central nervous system. In epidemic poliomyelitis in man, there are perivascular cellular infiltrations in certain areas. Hog cholera, although free from striking gross lesions, is accompanied, nevertheless, by important tissue changes.

Changes in the Central Nervous System

Careful, histological examinations of tissues from hogs with various bacterial infections (swine erysipelas, bipolar, paratyphoid and streptococcic infections) as well as those from hog cholera, were made by David and Rohrer,[7] who were unable to find changes in any of these diseases resembling those produced by hog cholera virus in the blood-vessels and the central nervous system. In the diseases examined, aside from hog cholera, the lesions in the central nervous system noticeably were absent. The complications (secondary infections) frequently found in hog cholera seem to have no influence in connection with the lesions in the central nervous system caused by the action of the filtrable virus.

Seifried[8, 9] found the characteristic changes in the central nervous system in 85 per cent, while Rohrer[5, 6] found them in 75 per cent of the acute cases of hog cholera. Thus, even if we should accept the hemorrhages and perivascular infiltration in the central nervous system as characteristic, we would still have approximately 25 per cent of the cases of hog cholera where such lesions are absent. This provides a serious handicap from the standpoint of diagnosis.

The lymph-nodes show lesions more constantly than the central nervous system. The changes here are primarily in the blood-vessels and are essentially of the same character as those described in the central nervous system. The parenchyma in many glands reveals hemorrhages here and there, and very seldom are the lymph-nodes free from histological changes. The sinuses of the lymph-nodes are usually free from erythrocytes. The changes in the lymph-glands undoubtedly account for the lymphopenia which is commonly present in hog cholera.

Changes in the Spleen

During the past two years, changes in the spleen have received increased attention, as a possible factor in the diagnosis of hog cholera. Rohrer and Heinz found that, in hogs inoculated with virus, 50 to 60 per cent will develop, particularly from the fourth to seventh day following injection, anemic and mixed infarcts which occur primarily near the border of the spleen. They represent in form and extent the area supplied by follicular arteries, which have undergone marked changes. These changes consist primarily of hyaline swelling so that the lumen is closed at the points of branching and constriction. The result is a coagulation necrosis of the white pulp. The more slowly an artery becomes occluded, the more marked is the development of the hemorrhagic marginal zone. Spleens from cholera hogs, showing no macroscopic lesions, reveal only a slight increase in pulp cells and here and there a perifollicular hyperemia. Delez[10] has studied the splenic lesions in cases of hog cholera in this country, and has found the same changes as were described by the European workers. Whether these lesions may be found in other diseases than hog cholera awaits determination.

In chronic cases of hog cholera, conditions are somewhat different and in some respects more difficult. While the changes in the central nervous system fail in only a comparatively few cases, the development of these lesions is much less marked than in acute cases. In the lymph-nodes, the regressive changes

in the blood-vessels, which are considered characteristic of acute hog cholera, are practically absent in the chronic form, but, in their stead, changes described as a perivascular fibrosis appear.

All of these studies have not furnished the rapid, yet reliable, method of diagnosis so much desired. If hog cholera virus is present in a herd, it is very important that it should be recognized immediately. Animal inoculation is too slow and expensive because pigs must be used as laboratory animals.

In making a field diagnosis, especially when immunization is being considered, we must still advise reliance upon petechiae in the lymph-nodes, kidneys and other organs, encephalomyelitis, and epidemiology. Pathological changes are more dependable than clinical manifestations.

Luttschwager[11] claims it is possible to make a differential diagnosis by careful histological study of the kidney. He claims to have found changes different from those in any other disease. His findings have not been verified.

Complicating Infections Confuse Diagnosis

In subacute and chronic cases, the complicating infections may mask the changes produced by virus infection, and since the lesions produced by the hog cholera virus are less conspicuous, one should therefore be all the more diligent in his search for them, when there is a possibility that hog cholera may be present.

Suipestifer enteritis, which may be a complication of hog cholera, is very common as a primary disease in the Corn Belt, and is sometimes difficult to detect clinically. Such herds sometimes show a high mortality when given the simultaneous treatment. All the complications which swine erysipelas may produce in our already complex situation will come to our attention as the incidence of the disease increases.

The work of Murray and Biester[12-14] has revealed that coccidiosis is another agent responsible for outbreaks of enteritis in swine, which, although not so common as the suipestifer enteritis studied by them, nevertheless should be kept in mind in attempting a differential diagnosis.

Recent studies of hog cholera, especially those of the histopathology, have provided a basis for an understanding of the lesions ordinarily found in hog cholera. Since we know that the virus attacks primarily the endothelium of the small arterioles and capillaries, producing a hyaline degeneration, it is more readily understood why petechiae are the characteristic

lesions of hog cholera, and, in uncomplicated cases, the only macroscopic change. Changes in the vessel walls in the central nervous system also explain the incoördination and other nervous symptoms so commonly seen in hog cholera outbreaks. Unfortunately, we have not been able to locate a lesion that is constantly present in a particular tissue.

In a series of pathological studies of hogs, inoculated with filtered virus, the following conclusions were drawn by Waldmann and David[15]:

1. Clinical symptoms are non-specific and a definite diagnosis is possible only after a pathological study.

2. During the period of 4 to 10 days following inoculation, the lymph-nodes most frequently show deviation from the normal. During the fourth and fifth days, hemorrhagic infarcts were found in the spleen in 50 to 77 per cent of the cases. The kidneys revealed hemorrhages in the cortex in 55 to 85 per cent of the cases. Necrosis of the solitary follicles may be observed in exceptional cases as early as the fifth day. Hemorrhages in the skin appeared after the fifth day. According to their observations, the first visible lesions may be found on about the fifth day.

3. The longer the course of the disease, the higher the ratio of complications or secondary infections.

4. A contagious pneumonic disease of swine (swine plague), if, in fact, it occurs at all, is exceptionally rare.

In closing, it is suggested that, although the histopathological and other studies of hog cholera which have been made during the past few years have not given us the decisive answers we desired to a number of questions, notably the one concerning a rapid, yet accurate, method of diagnosis, the work has been of great help to us in our understanding of the nature of the disease. Further work should be encouraged on all phases of swine diseases.

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DISCUSSION*

DR. J. W. CONNAWAY: I do not think we should let three important papers like these go by without at least some commendation, and you will agree with me that these papers are among the best we have ever listened to.

I want to say a word or two about each of these papers. Dr. Stange's paper dealt with researches, principally, but in his conclusions he emphasized one very important point of interest to practitioners, and that is, that in spite of all this research into the nature of hog cholera, it does not help much in practical diagnosis; and we must still depend upon gross lesions that have been known for many years, particularly the petechiae which occur in the several organs which he named. In this connection you will recall that in times past Dr. Kinsley has called hog cholera a *lesionless* disease, and I think it was his notion, when he used that term, that the petechiae, when present, were not necessarily related to hog cholera, but were primary or secondary lesions of bacterial origin; and they may to some extent be due to such cause. But for practical field diagnosis the petechiae must still be regarded as dependable signs of hog cholera. Nor should we forget our old teachings concerning hog cholera, namely, its infectiousness, its clinical symptoms, and even the gross lesions emphasized by the pioneer investigators, Dr. Salmon, Dr. Law and Dr. Detmers.

We of course know now that "button ulcers" are due to secondary infections; but in my experience—and I think in the experience of most of you—these lesions will be found so consistently associated with hog cholera outbreaks, that for practical diagnosis they are good presumptive evidence of the presence of that disease in the herd. In all hog-yards the secondary bacterial wound invaders are usually present, and when the filtrable virus makes a petechial lesion in the intestinal tract or in the bronchial tract, an opening thus is made, into which these usually harmless secondary invaders may enter and produce the larger gross lesions. So, in practical work, in sick herds where you find button ulcers, I believe that in most cases you will also find hog cholera. I may be wrong in that, but I think it is still a sufficiently good sign to make us suspicious of an invasion of the herd by hog cholera.

A word concerning Dr. Kinsley's paper: The doctor gave us a splendid sketch of the history of hog cholera in its one hundred years of existence in America. The outstanding points I wish to comment upon are the matters of controlling and getting rid of the disease. He mentioned that I believe hog cholera to be an eradicable disease. I believe this very firmly, and that practical measures will accomplish it. The methods which the doctor has presented in his paper will lead us toward that very desirable end.

He has mentioned this very important fact—that it will require some altruism on the part of the veterinary profession to reach that end. It is of course the duty of veterinary sanitary officials to keep that objective in view. If you who are practitioners will do the same in your work, helping to prevent and control this disease on the premises of clients; and if you will coöperate with state and federal agencies that are interested in the eradication of this disease, we will get rid of it, or at least be able to control it in a creditable manner.

Dr. Kinsley does not emphasize any more strongly than I would the

*This discussion followed the presentation of the foregoing paper by Dr. Stange, as well as that of two others already published in the JOURNAL: "A Century of Hog Cholera," by Dr. A. T. Kinsley, in the October issue, and "Recognition of the Most Important Infectious Diseases of Swine in the United States," by Dr. Frank Breed, in the November issue.

desirability of the control of virus. If I had my way about it, there are some veterinarians who would not be privileged to use virus, because some of them use it carelessly. In fact, I believe that I am one of the original serum-alone men, and I am still strong for it; since with serum alone and proper sanitary control measures we can eradicate cholera on limited premises. This has been demonstrated time and again. Moreover, it has been demonstrated to be effective also in larger areas, and I am confident that, by proper coöperation, it can be made effective even in statewide areas. I confidently believe the time will come when hog cholera will be declared officially an eradicable disease, and that effective federal and state measures will be put in operation to assure the fate for that disease which we know soon awaits Texas fever and bovine tuberculosis, namely, complete eradication.

The eradication of hog cholera means taking away from the veterinarian a big source of income, but in the next hundred years, or Century of Progress, that source of veterinary income will vanish, if the visions which Dr. I. K. Atherton and I have had come true. How can we best promote the end which every veterinarian in his heart knows to be for the best interests of the live stock producers, and the food-consuming public? The control of virus is one of the prime necessities toward that end.

I recall that on a previous occasion similar to this, I expressed the conviction, in the presence of Dr. Dorset, that he and his colleagues, who made the wonderful discovery of a means of immunizing swine against cholera, made a serious mistake when they turned it loose to the commercial world to manufacture and exploit. My conviction has not been changed that if the manufacture and distribution of hog cholera serum, and virus, had been kept exclusively under federal and state management, for use in the official control and eradication of hog cholera, vastly better progress could have been made than has been made. It may be that some day the federal government and the states will be compelled to take over that feature of this work in order to get rid of this disease. (I would remind those who hold up their hands in holy horror at the thought of state medicine invading private rights, that the control and eradication of contagious diseases is an official state function and duty, and not an inherent private right to be exercised for personal financial gain. This will be conceded when we think of foot-and-mouth disease, contagious pleuropneumonia, glanders, Texas fever or tuberculosis. Moreover, no live stock sanitarian who has given adequate study to hog cholera can see any justifiable reason for classifying and dealing with hog cholera in a manner so essentially different from the other diseases mentioned.)

In a proper system of live stock sanitation, the local veterinarians can work a wonderful good to their communities, to the live stock industry, and to themselves, by keeping the ideal of the eradication of this disease ever before them. Dr. Kinsley brought out very forcefully the enormous cost to the swine-raisers of the country in losses and vaccination costs. These ought to be saved, and I hope that the idealism and good sense of this Association will, before the next "Century of Progress" has closed, effect the eradication of hog cholera.

I must not slight the excellent paper of Dr. Breed. It deserves the serious consideration of every member of this Association, since it brings forcefully to our attention a swine disease new to America: swine erysipelas—the "Rouget" of France or "Rothlauf" of Germany. I have seen and studied this disease in Germany, many years ago, but as yet have not seen a case in America, and being "from Missouri," I confess that I have been exceedingly skeptical and slow to believe that this serious infection of European swine had been transplanted to this

country, and into a state where swine raising and feeding is not a major agricultural industry as compared with the more strictly Corn Belt area. But the evidence has accumulated so that in the veterinary ranks there should be no doubting Thomases. The pertinent question arises: Shall the American Veterinary Medical Association at its session in Chicago, in the year 2033, celebrating its hundredth anniversary of today's meeting, have on its program a paper entitled "A Century of Swine Erysipelas," which will read much the same as Dr. Kinsley's paper on hog cholera? That it has spread to every state in the Union, and has cost the swine industry untold millions of dollars in losses and vaccination costs? If that should happen, the reader of that paper will have good reason to brand the veterinary profession and the live stock sanitary boards of today—both federal and state—as imbeciles who were asleep at the switch. Let it, therefore, be our diligent duty to move with the utmost celerity and force to the task of confining swine erysipelas to its present area of infection, and to the task of eradicating it. If I were the Tsar of American agriculture, I would transfer Dr. Breed and some other talented veterinarians who are engaged in commercial laboratory pursuits to the more needed and more useful official public service of locating every focus of swine erysipelas infection in the country and eradicating the disease. It would be my particular care to see to it that they and their families were well fed.

DR. HENRY HELL: We have listened to three very fine hog papers. These men from the Corn Belt have had vast experience with hog diseases. I am glad that Dr. Stange made the statement that swine plague, or contagious pneumonia, occurred as such very rarely, if at all. Swine plague, or contagious pneumonia, has often been confused with the slower forms of hog cholera or with that form of hog cholera which sometimes follows the use of an impotent virus, and I am glad to know that we can eliminate that from our minds when we meet that condition.

Five times as many hogs were vaccinated in 1932 as in 1915, and the loss from cholera is about the same as it was then. That should bring home to every practitioner the fact that the work we are doing can stand a lot of improvement. The elimination of virus by vaccinated hogs brings back to my mind a conversation between Dr. Connaway, Dr. Gibson and myself, in Kansas City, in 1914. Following that conversation I used check pigs in every healthy herd of one hundred or more where simultaneous vaccination was used, over a period of some years. I found that no infection of the check pigs followed in healthy herds on clean premises. Therefore, the elimination of virus by a simultaneously treated hog is very slight, if it exists at all.

The restriction of virus seems to be about the greatest problem that confronts us today. Putting virus in the hands of the untrained farmer is the greatest mistake that was ever made in live stock sanitation. When we think that as little as 1/300,000 cc may produce an active form of the disease, the idea of putting virus into the hands of a layman, who does not know the nature of the product, is one of the greatest mistakes that can be made. Dr. Kinsley told you of the results of farmer vaccination, showing a 10 per cent greater loss than those following veterinary vaccination, which explains itself.

The infection of the premises by simultaneous vaccination is practically nil in healthy herds on clean premises, but do not guarantee not to infect premises where some of these other diseases are present, because you may sicken a hog enough for it to die with hog cholera, and when you do you infect the premises.

Careless use of virus is a great factor. Dr. Kinsley has pointed out to you how the virus might leak from the needle-puncture in the hog.

I want to say to you that that is nothing as compared with throwing the virus bottles against the rocks in order to hear them pop, and a lot of other careless practices that we are being told about throughout the country. People do not realize the importance of a living virus. They take it as so much cough medicine or something else—it is "just virus, that's all." They don't regard it as a living entity.

I want to say, further, that right at this time, there is quite a little hog cholera in my territory. Last year, due to economic conditions, a lot of cheap vaccination was indulged in. I have kept no exact figures on the subject, but it is safe to say that 65 per cent of the cholera existing in my immediate territory today is due as a direct result of premises infected by vaccination last year.

DR. PETER SIMONSON: I just want to add a few words to what has been said. We have listened to three splendid papers. We have heard a splendid discussion. We are all agreed, as sanitarians, that we want to eradicate hog cholera, if possible. The blame for our failure should be placed where it belongs. The practitioner in the field is not to blame. As sanitarians we should be ashamed to admit that the distribution and misuse of virus exists all over the country and that untrained hands are disseminating this infection. The fault belongs with our federal and state governments.

12th International Veterinary Congress
New York—August 13-18, 1934

Special Poultry Number of the Veterinary Journal

The *Veterinary Journal* (London) designated the October, 1933, issue the "Special Poultry Number," and devoted its pages to papers and abstracts of papers presented at the Fifth World Poultry Congress, held in Rome, in September.

While the papers in the special number deal largely with poultry problems in Great Britain, a paper by two Americans was included—that on "The Quantities of Vitamin A Required by Pullets for Maintenance and Egg Production," by B. M. Sherwood and G. S. Fraps, of the Texas Agricultural and Mechanical College.

Preliminary to the papers is an editorial urging members of the veterinary profession to take a more active part in the study and treatment of poultry diseases. The editorial says:

> The veterinary profession, by its training in animal diseases generally, should be in a position to advise on, and control, poultry diseases. But there appears to be a general lack of interest by the profession as a whole, with the result that the work is apt to be undertaken by others who cannot be in a position to do the work as competently as we ought to be. There is an opening of considerable value for veterinary surgeons in this field, and we feel that more training in poultry husbandry generally, and poultry disease in particular, should be found in the various veterinary teaching institutions.

Comment on this editorial would be superfluous.

STUDIES ON FOWL-POX VACCINATION*

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It has been known for the past few years that fowl-pox can be successfully controlled by the feather follicle inoculation method. Unattenuated fowl-pox virus has been used most commonly as an immunizing agent. The virus is inoculated in defeathered leg follicles on chickens between the ages of three and one-half and four months. The birds develop a severe reaction at the point of inoculation and a scab forms. The size of the scab depends upon the number of follicles inoculated. At the end of four weeks, this scab usually has dried and fallen away. Birds which have recovered from such an inoculation remain immune to fowl-pox for life.

There are certain disadvantages in the use of fowl-pox virus as an immunizing agent. Parasitized flocks may show serious reactions. This may be manifested by losses or a retardation in growth. Pullets which are vaccinated when they are laying usually will cease laying. It has been stated by poultrymen that vaccinated pullets do not begin egg production so early as pullets not vaccinated.

In general the value of placing fowl-pox-immune pullets and cockerels in the laying-houses is far greater than the disadvantages mentioned. Because of the reports concerning the use of pox viruses other than those obtained from chickens, these studies were started. The object was to learn whether or not some other pox virus would give as good results without any of the disadvantages mentioned.

COURSE OF POX VIRUSES ON CHICKENS

It has been demonstrated repeatedly that the chicken is susceptible to the pox virus obtained from pigeons. This is also true with regard to the cow-pox virus. That neither one of these viruses finds the chicken an entirely suitable host is clearly demonstrated by the type of lesion produced. When the virus of cow-pox is applied to defeathered follicles of the chicken, a mild reaction results. The follicles swell but subside without scab formation or any visible effect on the bird. Pigeon-pox virus applied similarly on chickens causes a large swelling of the fol-

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licles, which persists for a longer time than when cow-pox virus is used. A small scab may be formed at the follicle tip. No diffuse scabbing appears and recovery of the inoculated area occurs promptly without leaving evidence of the virus activity. The fowl-pox virus produces a severe reaction in the vicinity of the inoculated follicle. The follicle first swells. This swollen condition gradually spreads so that the entire inoculated area is involved. Scab formation occurs which eventually covers the affected follicles. After this scab has dropped off, a healed area generally remains. None of the pox viruses mentioned appear to spread to other feathered parts of the body. In some chickens, inoculated with fowl-pox virus in defeathered follicles, pox lesions may appear on the comb, wattles and face. This has been explained by some to be a result of the generalization of the virus. We should not lose sight of the fact that blood-sucking insects may be responsible for some of these cases.

The virus may enter the blood-stream, but it has been shown by Doyle[1] and others that it is not constantly present in the blood.

Table I shows the courses of cow-pox, pigeon-pox and fowl-pox viruses on chickens. These figures are the results of observations on many inoculated birds and show distinctly the comparative suitability of the chicken as a host for the three pox viruses.

TABLE I—*Course of pox viruses on chickens.*

	LESIONS APPEAR (DAYS)	LESIONS DISAPPEAR (DAYS)
Cow-pox virus (feather follicle)	3	11
Pigeon-pox virus (feather follicle)	2–4	16
Fowl-pox virus (feather follicle)	3–9	25–59
Fowl-pox virus (comb)	6–17	22–86

CROSS IMMUNITY BETWEEN COW-POX AND FOWL-POX VIRUSES

Four chickens were inoculated by the leg feather follicle method with cow-pox virus. Eight days after recovery, each one was inoculated with cow-pox virus on one leg and with fowl-pox virus on the other leg. The feather follicle was used for these inoculations. Bird 1 was found to be immune to the cow-pox virus but not to the fowl-pox virus. Bird 2 was immune to both the cow-pox and fowl-pox viruses. Bird 3 showed partial immunity to cow-pox virus but none to fowl-pox virus. Bird 4 died before a test could be made.

The immunity to fowl-pox virus found in one of the birds inoculated with cow-pox virus was probably not due to the inoculation. Although the spread of fowl-pox among the pens was not detected, it is quite possible that it did occur unnoticed or the bird might have been immune when obtained.

Four chickens were inoculated by the leg feather follicle method with fowl-pox virus and allowed to recover. Eleven days after recovery, they were inoculated on one leg with fowl-pox virus and on the other with cow-pox virus. All were found immune to fowl-pox but susceptible to cow-pox virus.

It was demonstrated that the immunity for cow-pox and fowl-pox could be produced simultaneously in the chicken. Four chickens were inoculated with both viruses, one on each leg. Four chickens also were inoculated with a mixture of cow-pox and fowl-pox viruses. The feather follicle method was used. Immunity to the two viruses was demonstrated by subsequent testing with the separate viruses.

These experiments show that cow-pox virus does not immunize chickens against fowl-pox. Neither does fowl-pox virus immunize chickens against cow-pox. Although cow-pox virus has been amply demonstrated by others to possess no immunizing value against fowl-pox, these experiments confirm such reports.

It has been claimed by Zwick[2] that the virus of fowl-pox can be modified by pigeon or rabbit passage. This modified virus was thought to have advantages as an immunizing agent over the fully virulent fowl-pox virus. The following experiments are concerned with attempts to pass fowl-pox virus serially on pigeons and rabbits.

Attempts to Adapt Fowl-Pox Virus to Pigeons

Two attempts were made to adapt fowl-pox virus to pigeons. The fowl-pox virus used produced typical lesions on chickens and was considered fully virulent. Fresh fowl-pox virus was collected from the skin of a chicken which had been inoculated seven days previously in feather follicles. Three pigeons (A, B and C) were inoculated with the fresh fowl-pox virus by removing feathers from the breast region and rubbing in the virus suspension. Pigeon A revealed a slight follicle swelling two days after inoculation. Six days after inoculation, the follicles were distinctly swollen but by the eighth day the swelling had practically disappeared. Pigeon B revealed only a slight follicle swelling for eight days following inoculation. Pigeon C gave a reaction similar to B.

Eight days after inoculation pigeon C was sacrificed. The follicles of the inoculated area were reddened but only slightly swollen. The affected skin was cut out and reduced to fine pieces by grinding with sand. A suspension in saline solution was made and three pigeons (D, E and F) and two chickens were inoculated with it. All inoculations were made into defeathered follicles. Neither pigeons nor chickens showed any evidence of a "take."

A second series of experiments was conducted along the same line. Fresh fowl-pox virus was obtained by sacrificing a chicken showing a good feather follicle "take." Chicken 1 and pigeons A and B were inoculated in defeathered follicles. Chicken 1 developed a good "take" in six days. Follicle swelling was observed in three days. Pigeon A showed a slight follicle swelling together with a redness three days after inoculation. This slight swelling increased in size through the fourth day. It then subsided, disappearing in about eleven days following inoculation. Pigeon B showed a follicle swelling with redness four days after inoculation. When sacrificed six days after inoculation, the follicles were still red and swollen. The skin from the inoculated area was ground with sand and suspended in saline solution. Chicken 2 and pigeons C and D were inoculated. Chicken 2 did not show a follicle reaction. Later, when tested for fowl-pox immunity, the bird was found to be partially immune. Pigeon C showed a slight follicle swelling at three, four and five days after inoculation. Pigeon D showed a similar follicle reaction.

Five days later, bird D was sacrificed and a virus suspension made as previously described. This virus skin suspension was inoculated on two pigeons E and F. The follicle reaction in both of these was slight. Four days after inoculation, pigeon F was sacrificed and the skin suspension inoculated on pigeons G and H. A slight follicle swelling occurred three days after inoculation. This swelling disappeared in two days. The four surviving pigeons of this series later were shown to be susceptible to pigeon-pox virus.

There is one fault with these two trials. We failed to test the skin suspensions from all inoculated pigeons on susceptible chickens. This would have indicated the virus survival or non-survival in these pigeons. There is no doubt that the inoculated pigeons failed to show a definite "take." We can offer our opinion with others that fowl-pox virus does not readily become adapted to pigeon tissues.

Attempts to Adapt Fowl-Pox Virus to Rabbits

In the first series, fowl-pox virus was obtained by using comb crusts from an inoculated chicken. All the rabbit inoculations were made by first shaving a small area on both sides of the abdomen. The skin was scarified with a sterile needle. One area was inoculated with the virus material plus saline solution and the control area with saline solution alone. When carborundum and saline solution were used to pulp the rabbit lesion, the control side was inoculated with carborundum and saline solution.

Fowl-pox virus was inoculated on the scarified skin of rabbit 1. Two days later, hyperemia was observed over the inoculated area. The control side showed healing. On the next day, hyperemia and a slight swelling of the inoculated skin were observed. On the fourth day after inoculation, the skin reaction showed signs of subsiding. The rabbit then was sacrificed.

The inoculated area from the skin of rabbit 1 was reduced to pulp. A chicken inoculated with this material developed fowl-pox. Rabbit 2 was inoculated with material from rabbit 1. A slight swelling and hyperemia over the inoculated area were noticed for five days following the inoculation. Rabbit 2 then was sacrificed and the inoculated area of the skin pulped. A chicken inoculated with this material did not develop fowl-pox. Later, this bird was shown to be susceptible to fowl-pox virus. Rabbit 3 was inoculated with material from rabbit 2. No hyperemia or swelling was observed over the inoculated area. Four days after inoculation, rabbit 3 was sacrificed. A chicken inoculated with material from rabbit 3 failed to develop fowl-pox. Later this bird was shown to be immune to fowl-pox virus. Rabbit 4 inoculated from rabbit 3 failed to show any skin reaction.

A second series was planned similar to the one just described. The first rabbit showed hyperemia and a slight swelling on the skin four days after inoculation. The rabbit then was sacrificed. A chicken inoculated from rabbit 1 developed fowl-pox. Rabbit 2, inoculated from rabbit 1, failed to show any skin reaction. The skin pulp from rabbit 2 was prepared four days after inoculation. This material failed to produce pox on a chicken which later was shown to be susceptible to fowl-pox.

These two experiments show that the virus of fowl-pox is not easily adapted to the skin of the rabbit. That the virus was kept alive on the skin of rabbit 1 in both cases is quite clear. The fact that rabbits 2 and 3 failed to show any evidence of the virus indicates that the virus did not actually propagate on rabbit 1.

The success obtained by other workers in this adaptation was due perhaps to conditions not present in these two trials.

Inasmuch as the experiments so far had failed to supply a substitute virus for immunization, attention was given to pigeon-pox virus. That pigeon-pox virus produced a mild "take" on chickens already has been shown. Its non-scab-formation quality and the fact that it does not disturb egg production or generalize are distinct advantages.

Doyle[3] states that pigeon-pox virus confers an immunity among chickens sufficient to prevent a serious natural attack by fowl-pox virus.

Zwick, Seifried and Schaaf,[2] together with Lahaye, have published information which rates pigeon-pox virus as a good immunizing agent.

Johnson[4] states that pigeon-pox virus vaccine is very satisfactory as an immunizing agent for preventing natural infection of chicken-pox among chickens.

Johnson[5] states that pigeon-pox confers immunity under natural conditions for at least one year.

Michael[6] failed to find that pigeon-pox virus vaccine conferred sufficient immunity to chickens which enabled them to resist natural infection with fowl-pox virus.

Experiments with Pigeon-Pox Virus as an Immunizing Agent Against Fowl-Pox

The pigeon-pox virus used in these studies was obtained from Dr. T. M. Doyle, Ministry of Agriculture Laboratories, Weybridge, Surrey, England. It has been shown here and by others that pigeon-pox virus produces a mild "take" on chickens. When inoculated in defeathered follicles, swelling appears in two to four days. This swelling soon becomes very prominent but disappears, on the average, around the sixteenth day. In some birds there is a slight scab formation on the follicle tips. This scab never enlarges and the "take" is always without scab. Pigeon-pox virus, when propagated on chickens, does not disturb egg production. This fact has been observed by all who have worked with it. Our experience with pullets and hens in heavy egg production simply adds to the accessible data.

The testing of the value of pigeon-pox virus as an immunizing agent against fowl-pox was divided into two parts. The first part consisted of testing the immunity produced in chickens by direct inoculation with fowl-pox virus. The second part consisted of testing the immunity produced in chickens by exposing them to active cases of fowl-pox.

Pigeon-pox virus was prepared by inoculating pigeons in defeathered follicles over the breast. The type of lesions on the pigeon does not produce very much scab material for drying Soon it was found that attempts to produce a dessicated form of virus were not only time consuming but laborious. The most convenient method found consisted of preserving the skin lesion in glycerin solution and making dilutions from this stock suspension for inoculation. In order to determine whether or not the concentration of the glycerin would have any effect on the virus, tests were made. Thin strips of pigeon skin lesions were placed in undiluted glycerin, in 50 per cent glycerin and in 20 per cent glycerin solution. The samples were kept in the ice-box at a temperature of approximately 7° C. The results are given in table II.

TABLE II—*Effect of various glycerin concentrations on pigeon-pox virus.*

GLYCERIN CONCENTRATION	STORAGE (DAYS)	RESULTS
Pigeon-pox lesion tissue plus undiluted glycerin	18	Suspicious lesion on inoculated pigeon; later test showed pigeon-pox immunity
	44	No lesion on inoculated pigeon; later test showed no immunity
	88	No lesion on inoculated pigeon; later test showed no immunity
Pigeon-pox lesion tissue plus 50 per cent glycerin solution	18	Good pox lesion on inoculated pigeon
	44	Good pox lesion on inoculated pigeon
	88	Weak lesion on inoculated pigeon; later test showed pigeon-pox immunity
	141	Weak lesion on inoculated pigeon; later test showed pigeon-pox immunity
Pigeon-pox lesion tissue plus 20 per cent glycerin solution	18	Good lesion on inoculated pigeon
	44	Good lesion on inoculated pigeon
	88	Good lesion on inoculated pigeon
	141	Weak lesion on inoculated pigeon

From this simple test, it is quite apparent that, when glycerin is used as a preservative, a 20 per cent solution of it is certainly desirable.

PIGEON-POX IMMUNITY—ARTIFICIAL EXPOSURE

PART ONE

In two experiments 21 eleven-week-old Single Comb White Leghorn cockerels were used. They were inoculated with pigeon-pox virus in the defeathered leg follicles. Sixteen days after re-

covery, they were tested with fowl-pox virus by inoculating the defeathered leg follicles. Two of the birds were found to be immune to fowl-pox. All of the others showed good "takes" varying not appreciably from the controls.

These experiments show clearly that chickens immunized with pigeon-pox virus will not resist feather follicle inoculation with fowl-pox virus. The two birds which resisted this exposure probably had been affected with fowl-pox. No evidence of its spread was detected among the birds, but it is thought that it occurred in these two cases without being observed.

Pigeon-Pox Immunity—Experimental Pen Exposure Part Two (A)

This experiment was conducted with S. C. White Leghorn cockerels 62 days old. They were divided into four groups and housed separately. Group 1 was inoculated with fowl-pox virus in defeathered leg follicles. This was done in the early part of June and all birds showed good "takes." Group 2 was inoculated with pigeon-pox virus in defeathered leg follicles. All birds showed good "takes" and had recovered by June 22. Groups 3 and 4 were held in a susceptible state.

On August 24, the first pen of experiment birds was assembled. They were placed in a 10x12 house with a yard attached. All birds at this time were 141 days old. The pen was made up of four different groups of birds: Fifteen birds from group 1, which had been inoculated with fowl-pox virus and allowed to recover; 20 birds from group 2, which had been inoculated with pigeon-pox virus and allowed to recover. Birds from both of these groups had been allowed a period of approximately 60 days from the time of recovery to the time when the experiment began. Nineteen birds from group 3 were added to the pen as fowl-pox susceptibles. Twenty-one birds from group 3 were inoculated on the combs with fowl-pox virus. All showed good "takes" and are referred to as group 4 in the experiment. This group furnished the means of exposing the other groups to fowl-pox virus.

Table III gives the data concerning the spread of pox among the various groups.

The fowl-pox immunes, comprising group 1, successfully resisted exposure to the disease. Three birds in this group died. Autopsy revealed only emaciation.

The pigeon-pox immunes, comprising group 2, resisted apparent infection with nine exceptions. These nine birds showed typical or suspicious lesions of fowl-pox on the head parts. Table

TABLE III—*Showing spread of pox among various groups.*

Days After Exposure	Group 1 Fowl-Pox Immunes			Group 2 Pigeon-Pox Immunes			Group 3 Non-Immunes			Group 4 Active Fowl-Pox Lesions		
	−	S	+	−	S	+	−	S	+	−	S	+
13	15	0	0	20	0	0	19	0	0	0	0	20
21	15	0	0	17	3	0	12	3	4	0	0	20
27	15	0	0	14	1	5	3	5	11	0	0	20
34	15	0	0	13	1	4	2	0	17	0	0	20
43	12	0	0	16	1	1	10	1	7	1	0	17
49	12	0	0	17	1	0	9	9	0	0	18	0

− = No fowl-pox lesions.
S = Suspicious fowl-pox lesions.
\+ = Positive fowl-pox lesions.

IV shows more clearly the severity and duration of the lesions as observed on these nine birds.

The pox lesions observed on birds recorded in table IV were located about the head parts. These lesions were small, did not spread, and disappeared sooner than in the controls. In order to establish definitely the fact that the lesions observed were due to fowl-pox virus, an examination of lesion material was made. Scrapings were made from each of five positive birds on the 27th day. This material, inoculated on separate cockerels, produced typical fowl-pox. Two fowl-pox-immune cockerels inoculated separately with scrapings from two of the positive birds resisted the infection. There is no doubt that the lesions observed on these pigeon-pox immune birds were due to the fowl-pox virus.

TABLE IV—*Severity and duration of pox on nine pigeon-pox immunes.*

Band No.	Days After Exposure				
	21	27	34	43	49
47	S	−	−	−	−
21	−	S	−	−	−
8	−	+	+	+	S
11	S	+	−	−	−
16	S	+	S	−	−
36	−	−	+	−	−
52	−	+	+	−	−
20	−	+	−	−	−
24	−	−	+	S	−

Group 3 of non-immunes was used to indicate the spread of the disease in the pen. All birds but two developed typical fowl-pox lesions about the head. These two birds developed suspicious pox lesions which failed to increase in size sufficient for a diagnosis. In general it can be said that the pox lesions in this group were more distinct, spread more and did not heal so quickly as in the pigeon-pox-immune group. In table V the severity and duration of pox in this group are recorded.

TABLE V—*Severity and duration of pox among non-immunes.*

BAND NO.	DAYS AFTER EXPOSURE				
	21	27	34	43	49
1	+	+	+	S	S
2	−	S	+	+	S
3	−	−	+	+	S
4	S	+	+	−	−
5	+	+	+	−?	S
6	+	+	+	−	−
7	−	S	−	−	−
8	−	S	+	+	−
9	−	S	−	−	−
10	+	+		−	−
11	−	+	+	−	−
12	−	+	+	−	−
13	−	S	+	+	S
14	−	S	+	+	S
15	S	+	+	−?	S
16	S	+	+	+	S
17	−	−	+	+	S
18	−	+	+	−	−

The fourth group which furnished a source of the pox virus in the pen all showed good active comb lesions throughout the experiment.

Seven days after the termination of the experiment, all birds were tested for fowl-pox immunity. This was done by inoculating fowl-pox virus in defeathered leg follicles. Not a single bird showed a "take." Susceptibility to fowl-pox virus could not be expected in three of the groups. In the pigeon-pox-immune group, some evidence of susceptibility to this severe exposure might be looked for. It is true that nine birds of this group had shown some evidence of fowl-pox. We know that pigeon-pox inoculation alone will not produce sufficient immunity to withstand such a test. Could it be that minute lesions of fowl-pox occurred

on these birds recorded as negative? We know that the birds had not been exposed to fowl-pox prior to the beginning of the experiment. The successful maintenance of the susceptible group attests to the fact that the virus of fowl-pox was kept confined to certain pens. It appears that the birds in group 2 recorded as negative were affected with fowl-pox in such a mild form as to evade detection by the ordinary physical examination.

Pigeon-Pox Immunity—Experimental Pen Exposure Part Two (B)

The second experiment was practically a duplicate of the one just described, the only difference being that the immunized birds had been allowed a period of 100 days from the time of recovery to the time when they were exposed. All cockerels used in this experiment were 191 days old when this pen was assembled. As in the previous experiment four groups made up the pen. The first group consisted of 17 birds immunized with fowl-pox virus; the second group of 22 birds immunized with pigeon-pox virus; the third group of 19 birds showing active fowl-pox lesions on their combs; the fourth group of 23 birds susceptible to fowl-pox virus.

The pen was assembled October 13 and continued for 64 days. The first group immunized with fowl-pox virus resisted the exposure. The third group inoculated on the combs with fowl-pox virus exhibited good pox lesions throughout the experiment. Scraping the lesions assisted in keeping them active. Table VI gives the results in group 2, the pigeon-pox immunes, and in group 4, the non-immunes.

About the 27th day following exposure, an outbreak of fowl cholera occurred in the pen. The infection was of the respira-

Table VI—*Results of exposure of pigeon-pox-immune and non-immune groups.*

Days After Exposure	Group 2 Pigeon-Pox Immunes			Group 4 Non-Immunes		
	−	S	+	−	S	+
21	15	4	3	0	10	13
29	8	4	9	0	0	19
36	9	3	8	1	0	13
49	13	2	3	5	1	7
64	18	0	0	12	0	0

tory type. Four birds were lost from group 2 and eleven birds from group 4. Although this infection did complicate the experiment somewhat, it is felt that it did not confuse the observations entirely. Table VI shows that the pigeon-pox immunes resisted the pox infection much better than did the non-immunes. As in the first experiment, the lesions on the pigeon-pox immunes were much smaller and healed much more quickly than in the non-immunes.

SUMMARY

Inoculation of chickens with cow-pox virus failed to immunize against fowl-pox.

Fowl-pox virus was not readily propagated on pigeons and rabbits.

Undiluted glycerin has a destructive action on pigeon-pox virus in pigeon skin lesions. A 20 per cent solution of glycerin seems to be a desirable concentration for preserving the virus in pigeon skin lesions.

The inoculation of S. C. White Leghorn cockerels eleven weeks old with pigeon-pox virus failed to protect them against inoculation exposure with fowl-pox virus.

The inoculation of 62-day-old S. C. White Leghorn cockerels with pigeon-pox virus failed to protect all of them against natural exposure to fowl-pox virus. The birds were allowed 79- and 100-day post-recovery periods. As compared with the non-immunes, there was some immunity produced against the fowl-pox virus.

There are two more phases of this work which should be continued and which will be as suitable flocks present themselves for study. One is the value of pigeon-pox virus when used in flocks which have not been troubled with fowl-pox but are liable to exposure. The other part is the value of pigeon-pox virus in heavily laying flocks in which fowl-pox has appeared. The evidence presented here shows that pigeon-pox virus does not produce as solid an immunity as does fowl-pox virus among chickens. That pigeon-pox virus may have some value in checking fowl-pox among mature laying chickens seems to be indicated in some field observations. Pigeon-pox virus as an immunizing agent against fowl-pox does have a place in a control program. It remains for us to learn how and when to use it.

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Veterinary Service

The office of the county veterinarian has again come under fire of city taxpayers, the attack being based upon the theory that the state will supply veterinary service to farmers and that employment of a purely county official is needless expense.

It is true that the state will supply such service. But the state veterinarian's responsibility is solely to Springfield and not to the farmer. He may ruthlessly condemn a herd of Winnebago cattle and answer for it not to the taxpayers of this county but to the voters of the whole state. His interest is not in the welfare of a community but in a very general way toward agriculture in the state as a whole, with an eye always to politics at the state capital.

This is not to say that there are not good men in the state veterinary service. But is seems logical that a man with local responsibility will take a more interested attitude in the well-being of taxpayers and voters who are his clients. His attention will be better focused, his service will be more immediate, and perhaps more careful.

The farm community, which pays a high proportionate share of taxes in relation to its actual money wealth, does not get very much from the county government, in any case. It seems dubious policy to attack an office which pays back many fold in added agricultural wealth to the general community; wealth which has a way of crossing arbitrary city and country lines and being exchanged over the counters of our stores.

Editorial in Rockford (Ill.) *Star.*

Grandparents

Two prominent veterinarians and their wives recently joined the ranks of grandparents. A son born to Mr. and Mrs. E. W. Freytag, on October 26, made grandparents of Dr. and Mrs. N. S. Mayo, of Highland Park, Ill. Mrs. Freytag was Miss Mary Lou Mayo. Thomas Ferguson Chalkley arrived in New York City, November 14. He made grandparents of Dr. and Mrs. T. H. Ferguson, of Lake Geneva, Wis.

BACTERIA AS FOOD FOR OESOPHAGOSTOMUM COLUMBIANUM LARVAE*

By R. E. REBRASSIER, *Columbus, Ohio*
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The life cycle of *Oesophagostomum columbianum* may be divided into three distinct periods, embryonic, larval and adult. The embryonic period begins in the sexual organs of the adult female and is completed in the open. The larval period develops partly in the open and partly within the host animal, while the adult period is passed entirely within the host.

The larval period is divided into four stages, the first two being passed in a free-living existence, the third commencing in the open and completed in the host, while the fourth is entirely parasitic. During their free-living existence in the open, the larvae feed actively, grow in size, undergo two moults and finally reach the infective stage. Under natural conditions this period of life takes place in the soil which has been contaminated with feces of an infected host.

The general belief has been that nematode larvae feed upon organic material in the feces. Until recently, the exact food which they utilize had not been determined. A few instances have been reported in the literature in which nematode larvae have developed in material other than feces.

Leuckhart[1] placed female nematodes containing viable eggs upon damp earth. Disintegration of the worm took place, the eggs hatched and the larvae developed into the infective stage. Lambinet[2] obtained ova from the uteri of female worms and placed them on agar plates. He stated that the eggs hatched and the larvae developed to the infective stage. It is impossible to tell from the description of his method what food was utilized.

Metcalf[3] successfully reared a plant parasite nematode in a sterile medium composed of fluid agar containing plant juices. However, the growth was better in agar in which there had been a heavy growth of bacteria before sterilization. Steiner and Heinly[4] showed that *Monochus papillatus* did not live satisfactorily on agar plates inoculated with bacteria but could be successfully reared in soil cultures if given other nematodes for food.

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McCoy,[5] in his experiments with hookworm larvae, produced evidence to indicate that living bacteria constitute the essential food utilized by hookworm larvae in developing to the infective stage. He showed that the larvae were able to grow on 22 species of bacteria, but the different organisms were not all equally suitable as food. There were three species of bacteria on which the larvae did not grow, and they also failed to grow on dead bacteria.

The studies of McCoy suggested that nematode larvae, other than those of the hookworm, may also feed on bacteria, and it was decided to study the food requirements of the larvae of *O. columbianum*.

METHODS

The method used in recovering nodular worm eggs from feces was the one described by McCoy.[5] It consisted in thoroughly mixing freshly passed feces from an infected sheep in a quantity of water. The mixture then was washed through copper-wire sieves. The filtrate was allowed to stand in a sedimenting tube while the eggs settled to the bottom. The supernatant fluid was poured off and the sediment transferred to a 50-cc centrifuge-tube. This sediment was repeatedly washed by centrifuging. When the supernatant fluid became clear, saturated salt solution was added to the sediment and the material again centrifuged. The eggs then were collected from the surface and were rendered free of bacteria by treatment with a 5 per cent antiformin solution in 10 per cent formalin for 30 minutes. The eggs then were washed several times with sterile distilled water and then were introduced into the experimental cultures.

The bacterial cultures consisted of 20 cc of ordinary nutrient agar, placed in 200-cc Erlenmeyer flasks. They were inoculated with the organisms 24 hours before the introduction of the parasite eggs, which allowed a good growth of bacteria to develop before the larvae were ready to feed. Control cultures were made to check the sterility of the ova. The bacteria used for the cultures were *Bacterium coli*, as McCoy had shown that hookworm larvae developed very satisfactorily on this species.

The method employed to determine the number of ova introduced into each flask and the number of larvae which developed to the infective stage was the same as that used by McCoy, which was as follows: The ova were suspended in sterile distilled water and the number per cubic centimeter determined. One cc of this suspension then was added to each of the *B. coli* cultures, the control broth cultures and the plain water cultures. The average

number of larvae which hatched in the plain water cultures was taken as the number of viable eggs introduced into each flask. The number of larvae which developed in the experimental cultures was determined by making a direct dilution count. Fifty cc of warm water was added and the flask agitated sufficiently to obtain an even suspension of larvae and still not break up the agar. The number of larvae in two 0.5-cc portions of the suspension were counted and the sum of the two counts multiplied by 50 to get the number present in the culture. As stated by McCoy, this method has the advantage over the Baermann isolation in that all stages of larvae may be observed, while with the latter method only the living larvae which migrate from the culture can be counted.

Experimental Data

The development of the larvae of *O. columbianum* was tested on pure cultures of *B. coli*. Ten cultures were used and approximately the same number of viable ova were placed in each culture. The larval counts were made 10 days after the ova were placed in the cultures, which allowed sufficient time for development of the infective stage. The results of these tests are given in table I and show that a large percentage of larvae developed to the infective stage in these cultures of bacteria.

Table I—*Development of O. columbianum larvae on B. coli. (Number of viable ova per culture approximately 5,000.)*

Culture	Infective Larvae	Developmental Stages		Dead Without Growing	Total Larvae Hatched
		Alive	Dead		
1	4,500	100	120	20	4,740
2	4,750	110	80	10	4,950
3	4,600	90	130	40	4,860
4	4,460	120	110	60	4,750
5	4,500	100	110	30	4,740
6	4,400	130	120	50	4,700
7	4,520	140	90	20	4,770
8	4,380	200	100	40	4,720
9	4,650	150	80	10	4,890
10	4,700	90	90	20	4,900

Approximately 5,000 viable ova were placed in each culture of *B. coli* and an average of the 10 cultures used showed that 4,546 larvae (90 per cent) developed to the infective stage.

The greatest number of infective larvae was found in culture 2, in which 4,750 (95 per cent) were counted. An average of

123 larvae underwent some development and were still alive; 103 developed to some degree but died, and 30 larvae hatched but died without development. An average of 4,802 larvae (96 per cent) hatched in the 10 cultures.

In order to determine whether larvae could feed and develop on agar, bacteria-free ova were introduced into agar cultures. Ten days later, these cultures were examined and it was observed that most of the ova had hatched but none of the larvae had undergone any development. Sterile ova, which were placed in physiological salt suspensions of *B. coli*, hatched and developed to the infective stage. These two tests indicate that it is the bacteria themselves that larvae utilize as food. These results correlate with those of McCoy, who found also that hookworm larvae did not grow in bacteria-free filtrates, in dead bacteria or in autoclaved fecal cultures.

The data derived from this experiment indicate that the larvae of *O. columbianum* utilize living bacteria as food in their development to the infective stage. This does not exclude the possibility of other substances serving as food for these larvae, but the results of this experiment and those obtained by McCoy with hookworm larvae indicate that non-living substances contribute little if any to the nourishment of larvae under natural conditions.

SUMMARY

1. The development of the larvae of *Oesophagostomum columbianum* was tested on pure cultures of *Bacterium coli*.

2. Eggs of the nodular worm, *O. columbianum*, obtained free from feces and rendered free from bacteria, hatched in agar cultures of *B. coli* and the larvae developed to the infective stage.

3. Eggs placed on sterile agar hatched normally but the larvae failed to develop to the infective stage.

4. The data derived from this experiment indicate that the larvae of *O. columbianum* utilize living bacteria as food in their development to the infective stage.

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BOVINE STERILITY*

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Sterility or infertility constitutes one of the most perplexing and important problems confronting the cattle-breeding industry. There is a great interest in this condition and this is growing continuously. It is evidenced by the fact that the subject is being studied by many investigators in various countries throughout the world. Though the subject of infertility received attention prior to the World War, it was pursued with greatly increased vigor after the close of the war.

Studies in sterility, including therapeutic and surgical measures for the relief of the condition, were stimulated greatly by the rapid increase in the pure-bred cattle population, the values of which quickly rose to stupendous heights. The necessity of investigating the field of sterility was greatly augmented also by the rapidly increasing incidence of Bang's disease. At the present time, with a greatly lowered valuation of cattle, and many of the larger herds having been reduced in numbers or dispersed because of falling prices, there remains a demand for a better knowledge of the subject. The breeders of pure-bred cattle and live stock farmers in general realize from an economic necessity that the efficiency of their herds must be increased.

The problems of infertility are probably less acute in beef breeds than in the dairy breeds, though the breeding difficulties met with in the raising of beef cattle are far from being minor ones. Sterility when viewed from a practical standpoint is of greater importance in the female than in the male. Sterility in the male is not uncommon and its importance should not be minimized. Sterility may be temporary or permanent and is classified also as physiologic and pathologic. The physiologic classification, in a broad sense, includes those forms known as nutritional or dietetic and functional or hormonic.

Sterility may be local and specific in character, involving one or more of the reproductive organs, or it may result from improper care and management. Reduced fertility may develop also as the result of dysfunction or incoördination of the endocrine system. The attention of the student of reproduction is being attracted constantly to the investigations being carried out in con-

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nection with the sex hormones. It has been shown that two hormones present in the anterior lobe of the pituitary gland control the periodicity of the ovarian cycle. The estrual hormone has been isolated and recently was standardized.

Certain functions of the corpus luteum are well understood, but more information relative to its hormones is necessary in order that their standardization can be effected. This applies also to the male sex hormones, the nature and function of which have not been demonstrated satisfactorily as yet.

Naturally occurring deficiencies of certain mineral elements have been shown to affect reproduction. The minerals most often involved are phosphorus and calcium and there are indications that a diet deficient in iron and copper may affect fertility. The effect of a low-calcium diet upon reproduction probably has been over-emphasized as it is not generally considered that calcium deficiency exists to any marked degree under natural farm conditions. In an experiment recently concluded at the Minnesota State Experiment Station, it was found that a low-calcium diet did not result in reduced fertility. Theiler,[1] of South Africa, in an article on "Phosphorus Deficiency and the Infertility of Cattle," is of the opinion that the rôle of phosphorus in the fertility of cattle is uniformly greater than that of calcium or vitamins.

The etiology of transient infertility of the South African range cattle was found to be a phosphorus deficiency. This type of sterilty is characterized by indefinite periods of anestrum due to suppressed ovulation. Theiler's findings were confirmed by the field investigations of Eckles, Palmer and Becker,[2] of Minnesota, who found reduced fertility to be one of the chief phenomena of cattle raised on phosphorus-deficient farms. Experiments designed to determine the relation of phosphorus to reproduction are now being conducted at the University of Minnesota. Results so far obtained indicate that a diet low in phosphorus is deleterious to normal reproduction.

Obesity and over-feeding are known to be detrimental to optimal breeding efficiency. It is well known that numerous cows of the various dairy breeds have become temporarily and occasionally sterile following the completion of an official test record for milk and butter-fat production. There seems but little doubt that intensive feeding of concentrates for early maturity and high milk yields may interfere seriously with normal reproduction. Today the various breed organizations have modified their requirements for official testing, and milking four times a day with extremely heavy feeding is no longer a common practice. These modifications have resulted in part due to the present eco-

nomic situation as well as the attempt to decrease the errors in reproduction.

Little or no information is available to show that cattle raised under present farm conditions are affected with sterility as a result of a deficiency in vitamins. Vitamin E is known as the vitamin of reproduction or the antisterility vitamin. Recent information gained in the study of vitamins shows that vitamins A and B also are concerned in reproduction. Certain selected cases of temporary sterility of a functional type, according to certain workers, may respond to the therapeutic application of vitamin E. The feeding of sprouted oats or the intramuscular injection of the ether extract of wheat germ has been used to supply vitamin E. The value of this form of therapy is suggestive and worthy of further investigation.

The Rôle of the Corpus Luteum in Sterility

Transient sterility due to indefinite periods of anestrum often is associated with dysfunction of the corpus luteum. It has been noted that ovulation may occur in the cow in the absence of estrum. This condition has been referred to as silent heat. Veterinarians have also experienced the situation in which the signs of estrum failed to appear following the manual removal of the corpus luteum in cows affected with anestrum. Examinations of such cases in approximately ten days following the operation, with few exceptions, will reveal the presence of a newly formed corpus luteum. We often have massaged the uterus of cows within four or five days following the removal of the corpus luteum and in which there had not been observed symptoms of estrum. In many of the animals the condition of heat was developed to the extent that service was performed without difficulty.

In order to determine definitely if ovulation is suppressed in anestrous cows due to a retained corpus luteum, frequent and regular physical examinations must be conducted over a period of time exceeding the diestrous period of the estrous cycle. I believe as a general procedure the veterinarian is justified in resorting to manual removal of the corpus luteum in cows affected with reduced fertility because of anestrum. The removal of the corpus luteum in such animals usually is followed by the signs of estrum within three to five days. Persistence of the corpus luteum most often is associated with pyometra and mummification of the fetus. It is also persistent in cases in which there is maceration of the fetus.

A typical persistent corpus luteum has been defined as one that gradually becomes embedded in the central portion of the

ovary. Strict adherence to this definition would prohibit one from classifying the more superficially located corpus luteum so often encountered in pyometra and fetal dessication as being persistent. The removal of the corpus luteum for the purpose of expelling the contents of the uterus in cases of pyometra and fetal dessication is a highly satisfactory method of treatment. Such corpora lutea are physiologically active, as is indicated by restored muscle contractility of the uterus and subsequent ovulation. The reason why removal of the corpus luteum in healthy anestrous animals results in the development of estrum has not been explained satisfactorily. Neither are we able to explain the phenomena that occur by the removal of the corpus luteum in cases of pyometra and mummification of the fetus.

A number of specific infections are recognized as causing sterility in cattle. Of these, *Bacterium abortus* Bang or *Brucella abortus* is by far the most important. This bacterium is a frequent invader of the gravid uterus, as well as the fetus and fetal envelops. Placentitis frequently associated with infiltration and edema of the chorion constitutes a common and important symptom of uterine infection. That such lesions are caused largely by the invasion of *Bact. abortus* is evident from the fact that the retention of the fetal membranes is a common and frequent condition in herds where Bang's disease is known to exist. *Bact. abortus*, though not directly responsible for many of the pathologic changes occurring in sterility, frequently paves the way for secondary invading microörganisms that under certain conditions find a suitable field for growth and multiplication, resulting in either temporary or permanent loss of fertility.

Pyogenic bacteria, namely staphylococci, streptococci, *Bacillus pyogenes*, and members of the colon group are the ones most often encountered. The various members of the above named group also may be responsible for the act of abortion with subsequent sterility in Bang abortion-free animals. Such abortions are known as non-specific and probably occur with greater frequency than is generally considered. It is well known that retention of the placental tissues is a serious menace to the health of the cow. Retention of these membranes retards the act of involution and thereby permits the absorption of the invading bacteria and their products. Temporary infertility due to various pathologic conditions often develops in cows having suffered with retention of the fetal membranes.

The control and elimination of Bang's disease markedly reduce the percentage of retained fetal envelops and increase the fer-

tility of the herd. All cases of retained fetal membranes must not be construed as being due to Bang's disease. The condition is quite common in cows having given birth to twins and often is encountered in certain non-specific abortions.

The presence of a protozoan disease affecting the genital tract of cattle, characterized by abortion usually in the early stages of gestation, and subsequent sterility due largely to cervicitis and pyometra, is now known to exist in certain parts of the United States. The protozoan is a trichomonad and has been classified as the *Trichomonas vaginalis* variety bovis. Emmerson,[3] in 1932, published a preliminary report on trichomoniasis in cattle, thus recording the first cases observed in the United States. More recently the disease has been found to exist in certain herds in Iowa, and a very interesting article under the heading of "Trichomonads Associated with Breeding Troubles in Cattle" has been published by McNutt, Walsh and Murray,[4] of Iowa State College.

Of the various pathologic conditions affecting the reproductive organs, inflammation is first in frequency and importance. The most common lesion occurring in sterility as the result of bacterial invasion is endometritis either acute or chronic. Cervicitis occurs very frequently being regarded by many as the most common lesion in sterility in which infection is the etiological agent. Cervicitis often assumes a chronic form and yields stubbornly to the various forms of treatment. In cervicitis and endometritis, the exudate that is comprised of mucus and bacterial products may have a very deleterious effect upon the motility and life of spermatozoa and in this way cause infertility by lessening the incidence of fertilization or, if fertilization results, successful implantation may be inhibited. Cervicitis, metritis, and cystic changes in the ovary frequently coëxist in the same animal. It is difficult to determine which may be the primary lesion.

Pyometra is a fairly common condition giving rise to reduced fertility in the cow. Hydrometra occurs with less frequency. Salpingitis and hydrosalpinx are not uncommon conditions and frequently accompany diseases of the ovary. Cystic degeneration of the ovary is a common condition, being by far the most frequent pathologic condition that affects the ovary of the bovine. The simple or retention cyst is the most common type occurring in the cow and originates usually in the Graafian follicle though they may originate in the corpus luteum. Retention cysts may develop when there is failure of the Graafian follicle to develop

normally and in which there is failure of ovulation. In this way the follicle becomes distended into a follicular cyst.

The most common type of cyst is the small multicystic form. Superficial follicular cysts, in the early stages, can not be distinguished from normal follicles. Large single cysts may be encountered and ovaries containing one large cyst with one or more smaller ones are frequently noted. The cysts are thin-walled when occurring superficially, but in the cysts deeply located in the ovary the walls are thicker and more difficult and, at times, impossible to rupture.

The etiology of cyst formation in the ovary remains unknown. Bacteriologic studies of cystic ovaries have not associated any one organism or group of organisms as common causative factors. The symptoms produced by cystic degeneration of the ovaries usually are those of nymphomania and a tendency toward sex transformation, the nature of which is so well understood that it would seem unnecessary at this time to describe.

Cystic degeneration of the ovaries often is concurrent with inflammatory conditions of the oviducts, uterus and cervix. The ovaries apparently are endowed with marked power of regeneration, as is often observed in the successful treatment of cows in which there may be cystic changes in one or both ovaries. Long-standing cases that are characterized by extensive physical changes should be given an unfavorable prognosis.

Sterility in the Male

It has been stated frequently by constructive breeders that the sire is more than 50 per cent of the herd. Breeders often will continue the use of a proven sire that has reached an advanced age. This practice in a measure is desirable and constructive breeding, but it may and often does result in a lowered breeding efficiency of the herd caused by reduced fertility on the part of the sire. The sire may also be rendered temporarily or permanently sterile as a result of injury to the sheath, penis or testes, or from neoplasms or specific infections of the various genital organs.

Orchitis in the bull is not uncommon, and often is caused by infection though it may result from trauma. *Bact. abortus*, when present in the testes, is capable of producing acute and extensive inflammation, characterized by degeneration, necrosis, fibrosis and abscess formation. Inflammation of the epididymis and seminal vesicles, due to the invasion of *Bact. abortus* and other microörganisms, occurs with less frequency.

The investigations of Donham, Simms and Shaw[5] have brought out new and interesting facts in the relation of the bull to the problem of reproduction in cattle. They found a definite relationship to exist between the microscopic picture of the semen and the percentage of conceptions following the services at which such specimens were collected. They further showed that it is advisable to conduct more than one microscopic examination of the semen in determining the fertility of the sire. The development of seminal histopathology by Williams and Savage[6] has created new interest in this subject. The practical application of their findings aids in the diagnosis and control of infertility.

The animal geneticist in recent years has shown the importance of genetic causes in relation to the problems of reproduction in domestic animals. A monograph recently issued by the Imperial Bureau of Animal Genetics of Edinburgh, Scotland, describes "The Physiological and Genetical Aspects of Sterility in Domesticated Animals."[7] The authors list the free-martin and the condition of hermaphroditism under the general heading of developmental sterility. These conditions arise as the result of abnormalities occurring during the developmental stages of the embryo, caused by irregularities of endocrine secretions. The discussion includes also the general subjects of species crosses, physiological and morphological abnormalities of the reproductive system explainable on the Mendelian scheme, and lethal factors. Examples of each subject are described clearly. The importance of this phase of sterility, in a clear and concise manner, is brought to the attention of those interested in the problems of reproduction. It is quite possible that the form of sterility, commonly known as "white heifer disease" may be explained from a genetic standpoint.

The perplexities of the problem of sterility are due in part to the multiplicity of etiological agents and we cannot hope to solve this highly important condition without just consideration of all phases concerned. Preventive treatment of sterility demands careful herd management and stable hygiene and the control or elimination of Bang's disease as well as other infections that may attack the reproduction organs. Surgical procedure continues to be the most satisfactory method for the treatment of individual cases. The therapeutic application of the extracts of certain endocrine glands and the employment of agents containing the antisterility vitamins are indicated in the nutritional and functional forms of sterility.

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AVA

The AVA celebrated its silver anniversary with a luncheon, at the Hotel Tracy, Philadelphia, on October 26, 1933. The AVA is in no way related to the AAA, or the NRA, or the TVA, but is a very close relation of the AVMA, having been organized shortly after the Philadelphia meeting of the AVMA in 1908. The AVA membership is comprised of wives of veterinarians in and around Philadelphia, who have been meeting together fairly regularly for 25 years. Those who attended the AVMA meeting in the City of Brotherly Love in 1927 may remember the group of ladies who appeared in Quaker costume during the convention. They were the AVA.

12th International Veterinary Congress
New York—August 13-18, 1934

Keeping Wild Turkeys "Wild"

The Illinois State Department of Conservation recently has undertaken an experiment to restore the wild turkey as a part of the hunting life in the State. An island in Horseshoe Lake, Union County, in southern Illinois, has been chosen as the spot on which to isolate a group of 100 wild turkeys so that they will be free of human contacts and grow up really wild. It is planned to leave the flock on the island for several years, in the hope that they will multiply rapidly, overrun the island and seek new quarters throughout southern Illinois. At the present time, Illinois has no open season for wild turkeys, and it will be several years before the ban is lifted, according to Charles F. Thompson, director of State Conservation.

NOTES ON THE EXPERIMENTAL TRANSMISSION OF BOVINE ANAPLASMOSIS IN FLORIDA*

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Anaplasmosis is a disease of great economic importance affecting cattle over wide areas of the United States. During the last few years, this disease has received considerable attention at the hands of various investigators and much of importance has been determined from these studies.

The economic importance of anaplasmosis is due to the high mortality rate and to the facts that affected animals which survive an attack are unfavorably influenced by long periods of indisposition and unthriftiness, and animals that have recovered and which show no microscopic or clinical evidence of the disease remain carriers of the infection, as proven by the infectivity of their blood on direct inoculation into susceptible animals, which produces the disease in a typical manner. It is economically important because of the ease of its mechanical transmission; and because certain widespread vectors are capable of feeding on a diseased animal in one stage of their lives and transmitting the infection to susceptible cattle in the next stage.

Since anaplasmosis, uncomplicated with other diseases of this nature, has been responsible for several instances of heavy losses in purebred herds of dairy cattle in the East Coast section of Florida, and in view of the fact that the disease has been diagnosed in central Florida, thereby indicating that the infection is widespread in the State, it was deemed important that the Animal Husbandry Department of the Florida Agricultural Experiment Station undertake an investigation of the possible modes of transmission of the disease from affected to healthy cattle under Florida conditions.

MATERIALS AND METHODS

In order to make practical field observations for epizoölogical evidence of possible transmitting agents and to perform experiments to the best advantage, the work is being conducted at a field laboratory erected especially for the purpose and located in Palm Beach County, Florida.

For the cattle experiments two units of four large separately

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screened isolation stalls, surrounded by a moat of oil, were tightly constructed on a large cement block, 50 by 160 feet, which served as the foundation and yard of the structures. Upon entering the screened halls leading into the buildings, one passes first into a tightly-closed vestibule where any intruder may be captured easily before proceeding to the screened stalls. Sloping cement floors are provided for the stalls and a system of pipes three inches in diameter imbedded in the cement floors leads from each stall through the wall over the edge of the cement yard for the purpose of conveying the drainage to receptacles at the ends of the pipes. The ends of the drain pipes are covered with brass gauze, except while the stalls are being washed, when the gauze is removed and the open end of a feed sack is tightly secured around the end to allow drainage during the cleaning process.

The virus animals used in these experiments were purchased locally and were infected by intravenous inoculations, using the blood from a known carrier of anaplasmosis. The susceptible animals used in the transmission experiments were obtained from the dairy herd of the Florida Agricultural Experiment Station at Gainesville. The infection has not been known to occur in this herd and the animals have proven to be highly susceptible to experimental anaplasmosis. These animals were rid of insect life and shipped to West Palm Beach, where they were transferred immediately in clean trucks to the experimental enclosures. After another careful examination, which showed that they were free from ectoparasites, the animals, before being put on experiment, were housed for observation under conditions as nearly insect-proof as practical for a time corresponding to the incubation period of anaplasmosis. A separate and thoroughly boiled clean needle always was used for each animal when collecting blood for examination and all thermometers were kept in an antiseptic solution except when they were actually in use.

Under the above conditions, control animals, fed, handled and housed in stalls with cattle that were critically ill and dying with anaplasmosis, have remained free from the infection in all cases.

Epizoölogical Observations

From field observations it was determined that certain blood-sucking external parasites occurred in the area under observation. Among the more important of these were various horse flies, stable flies, horn flies, mosquitoes, lice, and several species of ticks, including the Gulf Coast tick, *Amblyomma maculatum;*

the gopher-tortoise tick, *A. tuberculatum;* the American dog or eastern wood tick, *Dermacentor variabilis;* the black-legged tick, *Ixodes ricinus* var. *scapularis;* the brown dog tick, *Rhipicephalus sanguineus,* and the rabbit tick, *Haemaphysalis leporis-palustris.*

Since various of these ectoparasites have been seriously suspected of transmitting infectious diseases of domestic animals, preparations were made to collect them and study the possible relation that some of them might have in the transmission of anaplasmosis. The purpose of the present paper is to set forth further experimental evidence of the importance of certain vectors in their relation to the transmission of anaplasmosis as determined by these studies. In the early tests, horn flies, *Haematobia serrata,* and various species of mosquitoes collected from cattle on infected premises and confined in stalls where sick and susceptible animals were housed, failed to transmit the disease after a reasonable length of time. Recent work in the Florida field laboratory shows that the disease may be spread mechanically under favorable conditions by means of certain blood-sucking insects, including horse flies and stable flies. In view of these facts, work was undertaken to check on these findings.

Experimental Investigations with Blood-Sucking Flies

Experiment 1: The mechanical transmission of bovine anaplasmosis from a sick to a susceptible animal was accomplished through the agency of the horsefly, *Tabanus fumipennis* Wied. During the period from June 6 to 15, 1930, approximately 100 flies of this species were allowed partially to engorge upon an animal undergoing an acute attack of anaplasmosis, after which they were transferred immediately to a susceptible four-year-old cow (130), upon which they were permitted to complete their engorgement. On August 7, or 62 days following the first exposure, *Anaplasma marginale* Theiler was observed by microscopic examination of carefully stained blood-smears in cow 130. The percentage of infected erythrocytes gradually increased in the animal, reaching an average on the ninth day of 15 cells per 1,000 parasitized, after which they gradually diminished, and no further evidence of the parasites was observed after the fourteenth day of their first appearance.

The disease thus transmitted was of a very mild form, it was not accompanied by any clinical symptoms, and the only method of detecting the mild parasitic invasion was by frequent examinations of carefully prepared blood-films. On September 2, 1930,

cow 130 was injected intravenously with 15 cc of blood from a known carrier of anaplasmosis. She suffered no reaction, showing the development of a relative immunity (prémunition) known to occur following an attack of the disease.

Experiment 2: Anaplasmosis was transmitted mechanically also from a sick to a susceptible animal by means of the biting fly, *Stomoxys calcitrans.* During the period from May 28 to June 6, 1930, several hundred *S. calcitrans* were placed in a screened lantern-globe and were allowed to feed alternately on a clinical case of the disease and on a five-year-old susceptible cow (128). The feeding-jar was screened on both ends by means of finely woven bobbinet which permitted haustellation on the animals through different nets and avoided contacting susceptible cow 128 with a blood-stained screen.

Frequent microscopic examinations of carefully prepared blood-smears of cow 128 showed the presence of *A. marginale* in the erythrocytes at the rate of four parasitized cells per 1,000 on the 42nd day following the first feeding of these insects. The anaplasmata persisted in the erythrocytes for 33 days, reaching an average of 12 cells per 1,000 parasitized. The animal promptly recovered without exhibiting clinical evidence of the disease which could be attributed to the mild parasitic invasion. An intravenous injection of 15 cc of blood from a known carrier of anaplasmosis given cow 128 a few weeks later produced no evident reaction.

It was apparent in these cases that no definite biological relationship existed between the blood-sucking insects and the parasite, *A. marginale,* but that this organism was conveyed from sick to susceptible animals on the blood-soiled proboscides of these insects. The presence of numerous anaplasmata in the erythrocytes adhering to the mouth parts of the flies used in these experiments was demonstrated microscopically by carefully staining beak smears after haustellation on the sick animals, and this fact, together with the large numbers of flies used in the alternate feedings, constituted favorable conditions for transmission.

Investigations with Dermacentor Variabilis

In this country, Rees[1] has incriminated several species of ticks in the transmission of anaplasmosis and the present writer has had the opportunity of carrying out the following experiments with one of these.

Experiment 3: Under properly controlled conditions anaplasmosis was transmitted at West Palm Beach, Florida, in a fatal

form, to a five-month-old bull by means of the American dog or eastern wood tick, *D. variabilis*. The ticks were allowed to engorge in the larval and nymphal stages on diseased animals showing numerous anaplasmata in the erythrocytes and they transmitted the disease as adults after engorging on the susceptible animal. The ticks were reared in the following manner:

On April 28, 1932, an engorged adult female tick, *D. variabilis*, was obtained from a dog in West Palm Beach and placed for ovipositing on moist sand in a culture tube. After ovipositing and hatching had occurred, the resulting unengorged larvae were placed to engorge on an acute case of anaplasmosis known as bull 201. This animal was purchased locally and was given an intravenous injection of 50 cc of blood from a known carrier. Bull 201 reacted to the inoculation and died from the infection. The larvae in the meantime, having been confined on the scrotum by means of a small cloth bag, engorged upon and dropped from the diseased animal, after which they were collected and placed under conditions favorable for further development.

After molting to the nymphal stage and having shown signs of being ready to feed, the nymphs likewise were permitted to engorge upon an acute case of anaplasmosis which exhibited numerous parasitized erythrocytes on the date of placement. After engorging the nymphs were placed on moist sand in culture tubes and the resulting adult ticks emerged on December 23, 1932.

On April 15, 1933, four of these adult male and six adult female ticks, reared as outlined above, were confined on the scrotum of susceptible bull 493 UF. A small cloth bag was enclosed tightly around the scrotum for the purpose of confining and catching any ticks which might detach. Ten days later, four engorged female ticks dropped into the bag. All unattached ticks were removed. Table I shows data as recorded, giving detailed observations made on the case.

Experiment 4: This experiment on a nine-year-old cow was conducted under conditions similar to those described under experiment 3, and with adult ticks of the same species. These ticks were permitted to engorge in the larval and nymphal stages on acute cases of anaplasmosis. On April 28, 1933, four male and four female ticks, *D. variabilis*, were confined near the sixth coccygeal vertebra of cow 236 UF, by means of a cloth sleeve secured in position by means of adhesive tape. Seven days later, one female tick had engorged. The others remained several days without engorging and were removed. Observations on this case are presented in table II.

TABLE I—*Clinical symptoms and changes taking place within blood of bull 493 UF.*

DATE (1933)	TEMPERATURE		PULSE PER MIN.	RESP. PER MIN.	HEMOGLOBIN (DARE)	NO. CELLS PER CUBIC MM.		NO. INFECTED RED CELLS PER 1,000	REMARKS
	A. M.	P. M.				RED	WHITE		
6–5	101.0	101.8	45	30	95	5,999,000	7,900	16	Fifty-first day after exposure
6–6	101.8	101.4	50	45	90	6,000,000	8,000	25	Animal becoming gaunt
6–7	102.0	103.0	90	80	70	5,650,000	10,280	44	Diarrhea; off feed
6–8	103.8	102.6	90	115	65	4,980,000	15,840	80	Bodies marginal
6–9	102.7	103.0	84	80	45	3,090,000	18,080	100	Progressive anemia
6–10	105.2	105.4	90	50	44	2,530,000	10,680	250	Many red cells destroyed
6–11	105.8	106.2	80	80	30	1,920,000	5,680	350	Anisocytosis; poikilocytosis
6–12	101.8	100.0	80	100	12	1,520,000	5,600	500	Animal *in extremis*
6–13*	99.0	98.0	85	124	−10				

*Animal died at 4:30 p. m. Pathological lesions typical of acute anaplasmosis.

TABLE II—*Observations showing clinical symptoms and changes occurring in blood.*

DATE (1933)	TEMPERATURE		PULSE PER MIN.	RESP. PER MIN.	HEMOGLOBIN (DARE)	NO. CELLS PER CUBIC MM.		NO. INFECTED RED CELLS PER 1,000	REMARKS
	A. M.	P. M.				RED	WHITE		
6–6	101.2	101.4	40	45	103	5,090,000	8,500	40	Thirty-ninth day after exposure
6–7	101.8	105.2	60	106	95	4,230,000	8,540	60	Clinical evidence in afternoon
6–8	100.6	103.8	60	80	65	3,040,000	8,900	80	Clinical symptoms pronounced
6–9	104.4	106.2	80	80	60	1,430,000	12,120	250	Many cells contain two bodies
6–10	101.4	103.0	85	70	35	1,410,000	13,960	...	Cells destroyed; no bodies observed
6–11	102.3	106.0	80	74	25	1,350,000	10,860	...	Anisocytosis, poikilocytosis, etc.
6–12	105.6	99.0	80	150	−10	2,320,000	8,120	...	Many immature red cells in circulation
6–13*									

*Animal died at 6:30 a. m. Pathological lesions typical of acute anaplasmosis.

The above observations show that the tick, *D. variabilis*, may transmit a fatal form of anaplasmosis in Florida and that the disease is capable of being transmitted in this state under conditions similar to those which exist in other parts of this and foreign countries. In the case of the ticks there are ten known species occurring in various parts of the world which have been recorded as capable of transmitting anaplasmosis. Two of these species, namely, *R. sanguineus* and *D. variabilis*, which were incriminated by Rees, are known to occur in Florida, within the area which has been released officially from the quarantine of the North American cattle tick, *Boöphilus annulatus*.

SUMMARY

1. Anaplasmosis was transmitted to a cow and a young bull by adults of *Dermacentor variabilis* which engorged in the larval and nymphal stages on clinical cases of the disease.

2. The periods elapsing between exposure of the animals and the appearance of the protozoan parasites in the erythrocytes were 39 and 51 days, respectively.

3. Death occurred in the cow on the *sixth* day and in the bull on the *seventh* day following clinical evidence of the disease.

4. Anaplasmosis was transmitted mechanically in a mild form under favorable conditions by means of *Tabanus fumipennis* and *Stomoxys calcitrans*.

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Insanity in Caged Animals

A noted explorer says that 90 per cent of the wild animals caged in zoos are insane. This, he says, is due to the fact that they have no play activities to occupy their attention. He suggests toys, with which they may amuse themselves, as a way of keeping them sane.

THE CHEMICAL CHARACTER OF THE BLOOD AND URINE OF COLTS*

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As the authors had been successful in showing, by tests of the blood and urine, that the pregnancy disease of ewes is an acidosis, they were encouraged to apply similar methods to certain obscure troubles of colts.

Although central Kentucky has been recognized for many years as a district in which natural conditions are favorable for the raising of fine horses, during the past 40 or 50 years conditions and circumstances surrounding the raising of Thoroughbreds have changed in many ways, resulting in new and peculiar problems, and we must meet these problems with new and efficient clinical methods of investigation. At the present moment one method of forcing the development of colts, and preparing them for the race-track as two-year-olds, consists in feeding concentrated foods, administering haliver oil, and exposing them to violet rays. An authoritative statement that overfeeding may cause leg weakness in colts is not sufficient to check such faulty methods of feeding, and many of those responsible for the development and training of Thoroughbred colts insist on being shown just how overfeeding may injure the growing colt.

Examinations of the blood and urine of normal sheep, cattle and horses had indicated what might be taken as normal. However, as we were dealing with horses of a given age, it was felt advisable to run a check test on normal Thoroughbred yearlings. Because it is easier to catheterize them, four yearling fillies, of a group of six colts and seven fillies, were selected. This group of yearlings was considered, from conditions of breeding, feeding, care, development and health, as fine as any yearling colts in Kentucky. They had been fed not to exceed six or eight quarts of grain each, per day, with liberal hay and pasture. They also received, during the fall and winter, one teaspoonful of dicalcium phosphate each, per day, and were treated for parasites of the digestive tract every 60 days, from weaning on. Chemical determinations of the blood-serum calcium, potassium, phosphorus, sulfate S., non-protein nitrogen, and sugar were made. At the

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same time the urine was examined for calcium, sulfate S., ammonia and albumin, and the reaction and specific gravity were noted. Tables I and II show the results of one series of blood and urine analyses for each filly. The urine, obtained by passing a catheter, was examined within the hour.

TABLE I—*Analyses of blood serum of normal yearling fillies (mg in 100 cc).*

COLT	CA	K	P	NON-PROTEIN N	SULPHATE S.	SUGAR	CA/P
1	12.1	16.3	5.0	25.8	14.8	106	2.4
2	11.7	16.3	5.2	32.9	15.6	106	2.2
3	12.2	14.9	4.7	25.8	11.6	106	2.6
4	12.0	14.2	5.0	28.2	11.6	106	2.4
Average	12.0	15.4	4.9	28.1	13.4	106	2.4

TABLE II—*Analyses of urine of normal yearling fillies (mg in 100 cc).*

COLT	SP. G.	PH	ALBUMIN	NH_3	CA	SULPHATE S.
1	1033	7.6	0	50.0	80.9	77.9
2	1041	7.0	0	100.0	180.3	85.2
3	1035	7.2	0	20.0	177.5	78.2
4	1045	7.6	0	30.0	75.2	82.2
Average	1038	7.4	0	50.0	128.5	81.9

Thoroughbred colts raised in central Kentucky generally are fed too much grain. In a group of 37 Thoroughbred colts and fillies, eight animals showed clinical evidence of leg deformity which developed subsequent to weaning and manifested itself as the so-called crooked legs. The animals had been fed ten quarts each, per day, of a mixture of eight quarts of oats, one quart of corn, and two quarts of bran, and, in addition, received a tablespoon each, per day, of dicalcium phosphate. They also received haliver oil and, for several weeks during the winter, had been exposed to violet rays, using three lamps for three minutes daily. Each animal had received a one-half ounce capsule of carbon disulfide in October, 1932. On February 1, 1933, an examination of feces for parasite eggs indicated that all were heavily parasitized. The group was given carbon disulfid on February 18, 1933, and oil of wormseed on March 2,

1933. The animals (five fillies and three colts) which showed clinical symptoms were given a second treatment with oil of wormseed on May 16, 1933.

Tables III and IV show the results of one series of blood and urine analyses for each filly receiving concentrated foods, haliver oil, and exposure to violet rays. The urine, obtained by passing a catheter, was examined within the hour.

TABLE III—*Analyses of blood serum of overfed fillies (mg in 100 cc).*

COLT	CA	K	P	NON-PROTEIN N	SULPHATE S.	SUGAR	CA/P
5	11.9	19.0	5.5	23.6	9.8	121	2.2
6	12.9	19.7	5.0	16.3	14.4	121	2.6
7	12.5	18.3	5.0	16.3	7.3	139	2.5
8	12.8	17.0	4.7	23.6	8.5	113	2.7
Average	12.5	18.5	5.0	19.9	10.0	123	2.5

TABLE IV—*Analyses of urine of overfed fillies (mg in 100 cc).*

COLT	SP. G.	PH	ALBUMIN	NH_3	CA	SULFATE S.
5	1027	5.0	0	100.0	190.8	80.0
6	1037	7.8	0	100.0	278.8	50.1
7	1029	5.2	0	100.0	153.6	43.8
8	1050	8.6	0	100.0	116.4	78.6
Average	1038	6.6	0	100.0	184.9	63.1

The blood serum of the overfed fillies shows increased calcium and sugar, and diminished nitrogen and sulfur; while the urine also shows increased calcium, diminished sulfur, and, what is more important, markedly increased ammonia, indicating an overtaxed liver.

With the overfed colts and fillies the grain ration was reduced over half and all other supplementary food and treatment were discontinued. Within two months the blood and urine of the fillies examined had returned to what is considered normal, and all animals which had leg trouble were showing marked improvement. Tables V and VI show the results of blood and urine analyses eleven weeks after the animals were placed on a rational diet.

TABLE V—*Analyses of blood serum of fillies after treatment (mg in 100 cc).*

COLT	CA	K	P	NON-PROTEIN N	SULFATE S.	SUGAR	CA/P
5	12.4	21.0	6.5	25.8	9.7	141	1.9
6	12.3	21.0	7.0	23.3	7.0	100	1.7
7	12.7	21.0	6.5	25.8	6.0	113	1.9
8	13.1	21.7	6.5	25.8	13.8	94	2.0
Average	12.6	21.2	6.6	25.2	9.1	112	1.9

TABLE VI—*Analyses of urine of fillies after treatment (mg in 100 cc).*

COLT	SP. G.	PH	ALBUMIN	NH_3	CA	SULFATE S.
5	1040	6.4	0	30.0	89.7	79.8
6	1034	6.2	0	30.0	131.5	52.4
7	1036	7.8	0	20.0	100.0	72.7
Average	1037	6.8	0	26.7	107.1	68.3

During the month of April, 1933, the animals in this group (37 in number) made a noticeable and uniform gain in weight and, by the middle of May, with the exception of two or three animals, all clinical evidence of leg deformity had disappeared.

Anniversary Meeting of the Veterinary Medical Association of New Jersey

Plans for the fiftieth anniversary meeting of the Veterinary Medical Association of New Jersey, at Newark, on January 4-5, 1934, are going forward and a special program is being arranged for the occasion, according to the Secretary, Dr. J. G. Hardenbergh.

Dr. C. P. Fitch, president of the American Veterinary Medical Association, is to be the honor guest of the occasion. He is to speak on the A. V. M. A. at the Association dinner, on January 4, and on some phase of the control of Bang's disease, an January 5.

Other special features also are being planned, one of which will be a review of the work of the Veterinary Medical Association of New Jersey since its organization a half-century ago.

COW'S MILK PROTEIN HYPERSENSITIVITY IN A WALRUS*

By C. R. SCHROEDER, *San Diego, Calif.*
Zoölogical Society of San Diego

Walruses in captivity are not the rule. The only one to be found on public display in the United States is in the San Diego Zoo. "Marie," a Pacific Walrus (*Odobenus divergens*), was cap-

FIG. 1. Young female walrus "Marie" two days after arrival. No evidence of nasal catarrh. Conjunctiva and palpebrae normal. Skin smooth and covered with a fine coat of hair.

tured in the Bering Sea on an ice floe, July 10, 1932, by the crew of the yacht *Stranger* under the directorship of its owner, Capt. Fred Lewis, of the Zoölogical Society of San Diego. It was presumed that she was born in the month of May.

*Received for publication, August 7, 1933.

Canned milk, nursing bottles and nipples were a part of the ship's accessory supplies, inasmuch as the capture has been planned. A kindly devoted able seaman was presented with the adopted baby. Some unidentified ticks were removed from her head and nose. She occasionally suffered "minor eruptions," as they were described by the sailor, without unduly exciting the normalcy of her feeding routine. She was kept in a large tank on deck, supplied with running sea water, and fed five times daily, consuming two dozen 14½-ounce cans of unsweetened condensed milk adjusted to the standard of normal milk, heated to 105° F., the hot well temperature in the engine-room.

FIG. 2. Patient after four months confinement in the Zoo, taking milk from a standard quart bottle filled from a thermos jug. Minute papules and loss of hair in irregular areas on head are evident.

After six weeks of uneventful passage, she was delivered into our hands in August, together with instructions as to the feed she had been receiving. No change was made in her diet since she apparently was growing normally and the change from salt to fresh water, with further climatic adjustment from frigid North to sunny California, was sufficiently taxing. (See figure 1.) Kept out of doors in a large pool, she found the direct sun, without compensating cold ice beneath, too much for her and she immediately was placed in doors, with access to a small concrete pool supplied with running fresh water. Small eroding areas appeared on her head and flippers, with some bleeding of the

eroded surfaces, which was thought to be due to fly-bites. The flies were eliminated from her quarters. The condition persisted. Though we were unacquainted with the consistency of walrus feces, Marie's stool did not appear normal. Immediately on arrival, she experienced a mild conjunctivitis, followed by drooling and occasional difficult breathing, and accompanied by a clear mucous discharge. (See figure 2.)

The conjunctivitis was thought to be due to the fresh water pool. Sea mammals do not keep the cornea moist with conjunctival secretions and frequent movement of the lids. It has been our experience that occasional salt water baths will allay mild conjunctivitis. Marie forthwith was placed in salt water artificially prepared to meet sea-water hydrometer reading (1,023-1,025), approximately 4 per cent salt.*

The excessive salivary drooling was thought to be due to teething, as molar buds could be felt.

The abnormally liquid feces flaked with mucus was thought to be due to improper feeding. Much correspondence brought us feeding formulae used in Europe for the mature Atlantic walrus. The London Zoo had obtained young walruses past the nursing stage to which they unsuccessfully fed whale blubber. No authority could be found for the composition of sea mammal milk,† other than a suggestion that it was possibly high in butter-fat content. We altered Marie's diet, not too rapidly, from a 3.6 per cent butter-fat milk to a modified milk containing 12 per cent fat. There appeared to be some improvement, as the feces appeared smoother, not excessively flaked but firm. Inflammation of all mucous surfaces persisted, and the skin lesions took on the form of urticarial wheals. It was surmised that the salt

*Elementary Composion of Sea Water

Element	*Grams per Kilogram*
Oxygen	858.90
Hydrogen	107.98
Chlorin	19.34
Sodium	10.72
Magnesium	1.30
Sulfur	.90
Calcium	.42
Potassium	.38

Traces: Bromin, carbon, rubidium, boron, silicon, iron, nitrogen, fluorin, copper, phosphorous, silver, arsenic, lithium, iodin, thorium, zinc, gold, aluminum, barium, caesium, cobalt, lead, manganese, nickel, radium, strontium, vanadium. Salinity, 3.5 per cent; chlorinity, 1.937 per cent; H_2O, 96.5 per cent.

†On a recent Hancock Expedition to Guadalupe and Bonito Islands, a sample of seal milk was obtained. The following analysis was made by H. C. Bergeman and Dr. Eaton M. MacKay, of the Scripps Metabolic Clinic, La Jolla, Calif.

Single sample, California sea lion (*Zalophus californianus*):

Sp. gr., 1.016	Fat by extraction, 15.45
Solids, 31.9	Protein, 10.0
Ash, 1.07	Iodin number of fat, 95±

concentration was at fault. Her bath was small and the frequent change of water necessitated adding of salt which was not placed immediately in solution, so that the concentration varied rapidly, with NaCl the sole ingredient. This condition was remedied immediately. Skin scrapings were made to eliminate the possibility of scab mite or fungi involvement, though the lesions did not indicate either. Fecal smears did not demonstrate parasitic eggs or protozoa. The condition persisted. As molar teeth appeared, we added ground fresh salmon to the milk, with viosterol, cod-liver oil and calcium phosphate. (See figure 3.)

FIG. 3. At the height of a reaction an hour after feeding, five months after arrival. Almost total loss of hair, papular eruptions, mucous accumulation in nasal passages and oral cavity.

Various authorities indicated that the walrus fed on crustaceans at 10 to 20 fathoms. Others had seen them eating seal meat and one individual felt sure that he had seen them feed in the kelp beds not to the exclusion of meat or fish.

We were anxious to eliminate the milk at the earliest possible time. We felt that it was responsible for her occasional gastric distress, which at three different times necessitated administration of cathartics with a stomach-tube, and high enema. We had best success and greatest relief following administration of liquid petrolatum and castor oil given with a stomach-tube.[1]

The wheals on the body and flippers became immensely prominent immediately after feeding. There was scaling and bleeding of the mucosa of the anus and vulva and the skin adjacent was actively congested. The conjunctiva developed prominent granulations, the mouth was awry and drooling persisted. We felt that Marie, eight months in captivity, needed a change of environment. An effort was made to get her into the open in unadulterated ocean water on the beach with exposure to sun. Marie forthwith was transported to the Scripps Institution of Oceanography, at La Jolla. Through the courtesy of the director, Dr. E. G. Moberg, she was given the freedom of a 40,000-gallon tank on the beach in running sea water. Her milk intake was cut to a

FIG. 4. Two months after milk was discontinued. The palpebral thickening from long conjunctival irritation has persisted. Returned normal hair growth with no evident mucous membrane irritation.

minimum. After a trial for two weeks our patient showed no improvement. The skin was hot and dry. Erythema was generalized, the eroded areas were permanently fixed structures to remain as scars. Marie was returned to the Zoo.

Dr. Francis M. Smith, member of the Research Committee of the Zoölogical Society, suggested the possibility of cow's milk hypersensitivity. Marie was too squeamish and unhappy for us to attempt hyposensitization by the oral route. She was in need of immediate relief. Neither patch tests, intracutaneous injections of cow's milk or specific protein injections were made to gauge her sensitivity.

Walzer[2] states:

Milk acts as an offender almost exclusively as a food atopin in addition to asthma and cutaneous disturbances. It is a frequent cause of vasomotor rhinitis and gastrointestinal disorders. The active principle in the milk atopin is a protein. * * * Complete avoidance is the simplest method of treating milk sensitivity, as spontaneous loss of hypersensitiveness will usually take place eventually.

The reaction experienced was of equal degree regardless of thermal changes of the water in which she swam. The atopin here was unquestionably a milk protein, giving a constant reaction of equal intensity, whether raw, pasteurized or unsweetened condensed milk was used as part of the food.

FIG. 5. "Marie" at the present time, an exhibit in much demand, a popular performer in the hands of S. C. Charles, sea mammal trainer in the San Diego Zoo.

She was still receiving a small quantity of milk. It finally was eliminated from her diet. Immediate relief was experienced. The wheals failed to appear, the erythema cleared, the mucous membranes took on a normal appearance, and the conjunctival granulations were absorbed slowly. (See figure 4.) Connective tissue infiltration at the periphery of the lids probably will persist. Marie is destined to become an old walrus in the Zoo. She is easily handled and is eating chunks of local fresh fish covered with floured oyster-shell and cod-liver oil. She has the distinction of being a popular radio performer. (See figure 5.)

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[2]Walzer, M., Coca, A. F., and Thommen, A. A.: Asthma and Hay-Fever in Theory and Practice. (Charles C. Thomas, Baltimore, Md., 1931), p. 428.

FOWL LEUCOSIS*

By F. D. PATTERSON, *Ames, Iowa*
Department of Veterinary Investigation, Iowa State College

Subject: Barred Plymouth Rock pullet, S923.

History: Hatched, March 29, 1933; sire (B6411), normal; dam, C368E. Has shown some evidence of erythroleucosis since the breeding season. The spontaneous occurrence of leucosis in the flock from which pullet S923 originated has been noted very frequently. Myeloid, lymphoid-like, erythroid, nerve and eye

FIG. 1. Enormously enlarged liver of pullet S923.

types were represented. Fibrosis of leucosis lesions in this flock have been of comparatively frequent occurrence. An apparent transition of primary leucosis lesions toward fibrosis has been noted.

On April 14, 1933, pullet S923 was injected intraperitoneally with 0.5 cc of suspension V442. This suspension was prepared from affected tissues of a mixed type leucosis case.

August 8: Apparently in good health.

September 17: Apparently in good health, except for bruised-like condition over one eye.

*Received for publication, October 27, 1933.

October 12: Poor general condition. Emaciated to a considerable degree. Abdomen tense and full. General appearance of pullet is listless and dull.

October 13: Hemoglobin reading (Tallqvist), 60. Blood-smear indicated a mild erythroleucosis and myeloid leukemic condition. Killed for autopsy.

FIG. 2. Dissection showing involved thyroids, thymus and white myeloid neoplastic tissue at lower end of trachea of pullet S923.

Lesions: Generalized myelocytomatosis. In general the pathological tissue was very light gray, in many instances practically white, dull in appearance, and with a pinkish suggestion in some of the lesions. The liver was enormously enlarged (fig. 1) and

involved throughout, with diffuse myelocytomatous tissue, with many small, dull, white concentrated tumorous areas. The general color of the liver alternated between that of a very light gray and pink. The spleen was much enlarged and showed the same pathological involvement as the liver. The kidneys, bone-marrow, thymus, ovary, thyroids, parathyroids, some of the ribs, along the sides of some of the intercostal nerves, sternum and along its processes, pancreas and trachea at lower larynx also were involved (fig. 2). Muscle tissue of the breast and the mucous membrane of the intestine showed what appeared to be small, white myeloid-leucosis areas. Two small fibrous skin tumors were noted. Fresh stained (Wright) smears from the liver and thyroid tumor revealed a typical myelocytomatous process. Fixed sections have not yet been examined.

Diagnosis: Leucosis of the myeloid type.

Remarks: One of the most significant features of this case is the fact that the neoplastic tissue in this enormously enlarged liver belonged to the myelocytomatous type. Our research studies indicate that myelocytomatous lesions such as found in pullet S923 are caused by the same agent that causes the other types of transmissible leucosis. In our experience the majority of the enormously enlarged livers found in leucosis cases are lymphoid-like in character. In most instances the involved cells, for the greater part, are undifferentiated lymphoid-like cells which may possess potentialities toward either myeloid, erythroid or lymphoid types, this being governed by some unknown factor. Our studies indicate that all of these types are caused by the same transmissible agent. Therefore, their differentiation is of far less significance from a practical diagnostic standpoint than it would be if they were different disease entities.

12th International Veterinary Congress
New York—August 13-18, 1934

Doctor Ray Joins Corn States

Announcement has been made by the Corn States Serum Company, of Omaha, Neb., that Dr. J. D. (Jack) Ray will join that organization on January 1, 1934, and will be in charge of investigation and diagnosis, a new department created for the purpose of rendering a diagnostic service for practitioners. Dr. Ray has been a partner in the Kinsley Laboratories for about sixteen years. He will retain this interest and will continue to supervise the production of Kinsley biologicals as heretofore.

THE PHYSIOLOGY OF DOMESTIC ANIMALS. H. H. Dukes, Professor of Veterinary Physiology, New York State Veterinary College at Cornell University, Ithaca, N. Y. 391 pages, 218 figures. Edwards Brothers, Inc., Ann Arbor, Mich., 1933.

An urgent need for a modern, English text dealing with the physiology of the various domestic animals has existed for more than a decade and has constituted a problem of particular magnitude especially in the teaching of veterinary physiology. Although the appearance of Professor Dukes' book is particularly gratifying to those engaged in veterinary instruction, the student of comparative physiology, the veterinary practitioner, and the animal husbandman also will welcome this valuable source of reference. The title is in keeping with the generally accepted fact that a knowledge of functional phenomena is exceedingly important to all who are interested in the scientific and successful rearing and care of domesticated and also wild species of animals.

The fundamental value of mammalian physiology has been more keenly appreciated during recent years and in consequence extensive experimental study has been carried on in this field with a rapid accumulation of knowledge resulting. Although a better knowledge of human physiology has been the main objective, the experimentation has been conducted to a large extent with small domestic animals suitable for use under laboratory conditions and a great amount of physiological data has been made available for these species. The large domestic animals have received but limited experimental attention, as the author notes in his preface, and authentic data pertaining to functional reactions in the horse, ox, sheep and pig are therefore lacking to large extent.

The author has arranged the subject matter in eleven parts, which in turn are subdivided into 39 chapters.

An introductory chapter deals with a discussion of physical and chemical phenomena concerned in physiological activity of body tissues. The first chapter takes up a description of the functional characteristics of blood and lymph and in the succeed-

ing chapters the various systems of organs, muscle and nerve tissue and metabolism are given consideration. Part eleven is devoted entirely to the physiology of reproduction.

Illustrations, graphs and tables are freely used and add greatly to the descriptive data.

Experimental facts, so essential to authoritative scientific knowledge, are constantly referred to in connection with every phase of the subject with names of investigators making the particular contributions. A comprehensive list of references also is appended to each of the eleven parts, thus greatly facilitating a more detailed survey of the literature.

The arrangement of subject matter contributes to a high degree of flexibility of the book as a teaching text, as study can be begun with any one of the eleven parts without confusion. This is an essential attribute, as few teachers agree upon the order in which the various divisions of a subject be presented.

The author has made a very excellent selection of those phases of the subject entitled to the greatest consideration as regards practical value. This feature will be appreciated by the veterinary practitioner who uses sound physiological facts for his diagnostic and prognostic reasonings.

It may be remarked before closing this brief review that the clarity and conciseness of expression achieved by the author and his collaborators throughout the book will be found pleasing to readers.

Professor Dukes is highly deserving of the gratitude of the veterinary profession for his spirit and effort in contributing this valuable text to the literature. R. S. A.

THE PRACTICE OF VETERINARY MEDICINE. D. H. Udall, Professor of Veterinary Medicine and Superintendent of the Ambulatory Clinic, New York State Veterinary College at Cornell University, Ithaca, N. Y. 267 pages and 81 illustrations. Published by the author, Ithaca, N. Y., 1933. Cloth, $5.00.

This book is one of the most important which has appeared in American veterinary literature for a number of years. The author has been the Head of the Department of Veterinary Medicine at Cornell University since 1908, and during this long period of service has collected a wealth of data which heretofore have not been made available to the practitioners of veterinary medicine of this country and abroad.

The Ambulatory Clinic of the Veterinary College at Cornell University has furnished a large amount of material for study

and research. The author has made the most of this opportunity and has carefully investigated many of the problems with which the practitioner of veterinary medicine finds himself confronted. In our judgment, the author's discussions of disorders of metabolism, diseases of young calves, and chronic mastitis are outstanding.

The treatment of the subject is logical. Diseases of the respiratory system, digestive system, kidneys and other organs, are first. The infectious disease are next, first the acute infections and then the chronic infections. These are followed by a short account of the protozoan and metozoan infections. The last chapter is devoted to a discussion of poisoning. This publication is devoted to large animals and makes no pretense of being a treatise on the diseases of pet animals.

The size and arrangement of the book are unusual. It is printed on an excellent dull finish paper with a type that makes easy reading. The page is large and contains two columns. There are those, undoubtedly, who will not like this format. The illustrations do not appear as sharp as they would on glazed paper.

The author has made a careful study of the literature and has drawn freely upon the latest scientific observations and discoveries. This, added to the extensive personal experience of the writer and the records of the Ambulatory Clinic, has made it possible to present a valuable addition to our veterinary literature. The style and arrangement are excellent. Veterinarians, especially practitioners, should not fail to read this book. Further we might suggest that practitioners of human medicine would gain some valuable information from a careful study of this work.

W. L. B.
C. P. F.

Corn Belt Servitor

Under the name, "Corn Belt Servitor," another veterinary house organ made its bow recently. The first issue commemorated the twentieth anniversary of the Corn Belt Serum Company, Inc., of East Saint Louis, Ill. Dr. C. L. Campbell, who joined the company the first of the year as laboratory and field consultant, is editor of the new publication, which is characterized by several new departures in format and make-up. The policy of the *Servitor* will be "to afford some beneficial scientific material, a little fraternal and social fragrance, a slight diversion from toil, and to present suggestions of a unified sales service."

THE DETECTION OF PROTEINS OF THE NEMATODE HAEMONCHUS CONTORTUS IN THE SERA OF INFECTED SHEEP AND GOATS. John E. Stumberg. Amer. Jour. Hyg., xviii (1933), 2, p. 247.

Haemonchus protein was detectable in dilution of 1:50,000 by means of what is essentially an anaphylactic test. The test consisted of a rise in the relative percentage and absolute number of eosinophilic leucocytes in the circulating blood following injection of extract into sensitized guinea pigs. Injection of anti-haemonchus immune rabbit serum into unsensitized guinea pigs previously injected with Haemonchus extract caused a rise in percentage of eosinophils as long as 17 days after the injection of the extract. Serum of a helminth-free sheep, of a sheep infected with nematodes other than Haemonchus, and of a sheep following loss of Haemonchus infection failed to cause a rise in percentage of eosinophils in sensitized and control guinea pigs. The authors conclude that Haemonchus proteins are absorbed through the alimentary tract into the circulating blood of heavily infected sheep and goats in sufficiently unaltered form to react with specific antibodies in an anaphylactic test.

RESISTANCE TO INFESTATION WITH TRICHINELLA SPIRALIS IN HOGS. The eosinophilic and precipitin response. Geo. W. Bachman and Rafael Rodrigues Molina. Amer. Jour. Hyg., xviii (1933), 2, p. 266.

Total leucocytic counts, differentials, precipitin tests, and estimation of hemoglobin percentages were done in 10 young hogs for 345 days at 5-day intervals. Animals were fed 500 to 600 grams of trichinous meat every 80 and 40 days. Three biopsies were performed previous to each feeding and the average number of larvae in 20 to 30 microscopic fields was determined. In the five animals that survived there were a decided eosinophilia (highest 32 per cent) and high (1:6,000) precipitin titres during first infestation, but a rapid decrease of eosinophils and a total absence of precipitins in subsequent infestations. Convalescence

during repeated infestations was not correlated with high eosinophilia and precipitin titre. Immunization with intramuscular injections of variable amounts of 1 per cent suspension of trichina powder in Coca's alkaline solution produced an eosinophilia (16 per cent) and a high precipitin titre (1:6,000). Immunization previous to infestation afforded no protection against trichiniasis. It seems that the resistance to hyperinfestation is due to a retention of antibodies and a local mobolization of leucocytes and cells of the reticulo-endothelial system in the intestinal wall of the host.

EPIZOÖTIC FOX ENCEPHALITIS. IV. The intranuclear inclusions. R. G. Green, M. S. Katter, J. E. Shillinger and K. B. Hanson. Amer. Jour. Hyg., xviii (1933), 2, p. 462.

Intranuclear inclusions occur in the endothelial cells of foxes dying from epizoötic fox encephalitis They are found consistently in foxes dying of natural infection and after experimental inoculation. Similar inclusions are not found in foxes dying from natural infections with the distemper virus of Laidlaw and Dunkin. Inclusions develop in the endothelial cells of the central nervous system after experimental inoculation of fox encephalitis virus by intraperitoneal, intramuscular, and intratesticular injection, but with these experimental infections they do not occur in the ependymal cells. In experimental infections following inoculation by cisterna puncture and by intracranial injection where the virus is injected into the ventricles, intranuclear inclusions are found in the ependymal cells as well as in the endothelial cells of the brain and spinal cord. The occurrence of intranuclear inclusions in both natural and experimental infections, the absence of these inclusions in other infections, and the production of inclusions in the ependymal cells only when the virus is introduced into the cerebro-spinal fluid demonstrate that those intranuclear bodies are characteristic of the fox encephalitis virus.

VACCINATION WITH HEAT-KILLED AND FORMALIZED TUBERCLE BACILLI ON EXPERIMENTAL TUBERCULOSIS. Robert M. Thomas. Jour. Exp. Med., lviii (1933), 2, p. 227.

Rabbits vaccinated with tubercle bacilli killed by exposure to formalin (0.4 per cent) did not show any acquired resistance to subsequent infection with bovine tubercle bacilli; white rabbits vaccinated with tubercle bacilli which had been killed by heating to 70 degrees for 1 hour survived more than half as long again

as their controls. Intraperitoneal injection of either vaccine into guinea pigs made their skin sensitive to tuberculo-protein. The ratio of absorption when both vaccines were placed under the skin were the same. Following the intravenous injection of heat-killed tubercle bacilli, it was found that rabbits developed a massive tubercular pneumonia. The production and ultimate absorption of the cellular exudate, however, showed that these processes were similar to those found after injection of living bacilli. The lesions which followed injection of heat-killed organisms differed from those found in active tuberculosis in that in any one animal they showed a striking uniformity in appearance, while in the active disease the lungs showed a great diversity in type of lesion.

STUDIES ON PSEUDORABIES (INFECTIOUS BULBAR PARALYSIS, MAD ITCH). I. Histology of the disease, with a note on the symptomology. Weston E. Hurst. Jour. Exp. Med., lviii (1933), 4, p. 415.

The histology of pseudorabies differs materially in various animal species. In the rabbit, subcutaneous, intradermal or intramuscular inoculation leads to local inflammation and necrosis. The infection ascends the peripheral nerve to the corresponding spinal ganglia and segments of the spinal cord, where primary degeneration of nerve and glial cells take place. The nerve cell changes are probably responsible for the cardinal symptoms of the disease—itching. Death ensues soon after the virus reaches the medulla, before visible changes have been produced here. Intranuclear inclusions bearing some resemblance to those in herpetic encephalitis, yellow fever, etc., occur in cells derived from all embryonic layers. The disease in the guinea pig resembles closely that in the rabbit and is modified only by the slightly greater resistance of the animal. In the monkey, after intracerebral inoculation, widespread degeneration and necrosis of cortical nerve cells are accompanied by the appearance of specific nuclear alterations in nerve and glial cells, but not in cells of mesodermal origin. No lesions are found in other viscera. In the spontaneous disease in the cow, lesions approximate more closely those in the monkey than those in the rabbit. In the pig, vascular and interstitial lesions predominate. Nerve cell degeneration is relatively slight and typical inclusions are not observed. These differences probably explain the benign course of the malady following subcutaneous inoculation in this animal. The lymphatic system also participates in the reaction to the virus.

THE RELATION OF VITAMIN C DEFICIENCY TO INTESTINAL TUBERCULOSIS IN THE GUINEA PIG. Mack McConkey and David T. Smith. Jour. Exp. Med., lviii (1933), 4, p. 503.

Seventy-two adult guinea pigs were fed tuberculous sputum daily for periods ranging from 6 weeks to 4 months. Thirty-seven of these were maintained on a diet partially deficient in vitamin C; 26 developed ulcerative intestinal tuberculosis. In the remaining 35 animals, whose diet was supplemented by an adequate amount of vitamin C, only two developed tuberculous ulcers in the intestines. The ingestion of tubercle bacilli by the guinea pig is not the sole factor in the production of intestinal tuberculosis. An adequate supply of vitamin C usually protects the guinea pig against ulcerative intestinal tuberculosis.

THE SIZE OF THE VIRUS OF FOWL PLAGUE ESTIMATED BY THE METHOD OF ULTRAFILTRATION ANALYSIS. W. J. Elford and C. Todd. Brit. Jour. Exp. Path., xiv (1933), 4, p. 240.

The size of the elementary virus particles of fowl plague, as they exist in the body fluids of the fowl, has been estimated by ultrafiltration analysis, using graded collodion membranes, to be 60 to 90 millimicrons. Control experiments in which Staphylococcus K bacteriophage (size 50-75 millimicrons) was admixed with the virus suspension confirmed the close relationship in size between this phage and the virus. Differentiation of their filter abilities was only just possible.

FILTRABLE VIRUS CARRIERS. Charles S. Gibbs. Jour. Inf. Dis., liii (1933), 21, p. 169.

The author reports filtrable virus carriers in hog cholera, rinderpest, and infectious laryngotracheitis of poultry. This is the first instance in which filtrable virus carriers have been identified *a priori* by laboratory tests and not merely referred to as possibilities through *a posteriori* reasoning. Experiments in the transmission of hog cholera and rinderpest by carriers were conducted by the author in China.

THE EXCRETION OF URINE IN THE DOG. VII. Inorganic phosphate in relation to plasma phosphate level. Robert F. Pitts. Amer. Jour. Physiol., cvi (1933), 1, p. 1.

In normal dogs in which inorganic phosphate has been injected intravenously, the phosphate clearance is a curve linear

function of the plasma concentration. At low plasma concentrations the urine is phosphate free, but as the plasma concentration is raised the phosphate clearance rises and approaches the xylose and sucrose clearance without ever exceeding the latter. This relationship does not hold in phlorizinized dogs in which the maximal phosphate clearance is depressed below the level of the simultaneous xylose, glucose, and creatinine clearance for unknown reasons.

ENDEMIC PARATYPHOID INFECTION IN TURKEYS. Leo F. Rettger, Wayne N. Plastridge and Ruth Cameron. Jour. Inf. Dis., liii (1933), 2, p. 272.

A paratyphoid-like organism was isolated from turkey poults of various ages which were sent to the laboratory from two widely separated turkey farms. Large losses were sustained by the owners through annually occurring epizoötics which affected the young turkeys, particularly the very young poults. A general systematic study, including the application of the agglutinin-absorption test, established the organism as being of the *Salmonella aertrycke* type. It was highly virulent for young turkeys and for chicks, mice and guinea pigs when administered orally. The history of the two turkey farms and the enzoötics gave strong indication that the disease had been brought to farm B through small poults which were purchased from farm A. The mortality on both farms was very high.

CUTANEOUS PASSIVE IMMUNIZATION AGAINST TETANUS. A. Urbain. Abst. Arch. Path., xvi (1933), 1, p. 144.

Favorable results were secured in the prevention and cure of tetanus intoxication or infection in guinea pigs. Tetanus antitoxin of a potency of 3,000 units per 10 cc, was mixed with a petrolatum and hydrous wool-fat base. The ointment was applied to the shaven and slightly scarified skin of test animals between 24 hours preceding the injection either of toxin or of spores mixed with Staphylococci and three hours after such injections. This afforded a fair degree of protection; the action was specific.

SPONDYLITIS OF SWINE ASSOCIATED WITH BACTERIA OF THE BRUCELLA GROUP. William H. Feldman and Carl Olson, Jr. Arch. Path., xvi (1933), 2, p. 195.

A specific spondylitis of swine associated with bacteria of the genus Brucella was found in 24 different animals. Organisms

of the Brucella group were secured by culture and by inoculation of animals from them. The identity of the respective cultures was determined by their behavior in experiment animals and by their antigenic properties. The affected swine were apparently symptomless and without lesions in other parts of the body. The lesion was an encapsulated abcess-like structure occupying an irregular cavitation in the body of the vertebra. The disease exhibited an apparent predilection for the vertebrae of the lumbar and sacral region, and adjacent vertebrae were frequently involved. The majority of the specimens obtained were from animals less than one year of age and a relationship of the lesions of the vertebrae to the usual lesions of infectious abortion in swine was not observed.

New York Broadcasting Program

Under the auspices of the Committee on Broadcasting, New York State Veterinary Medical Society, the following talks were given over Station WGY (General Electric Station) from August 25 to November 17:

August 25—"Growths on Farm Animals," Dr. P. Olafson, Ithaca.

September 8—"Relationship Between the Veterinarian and the Farmer," Dr. M. C. Thompson, Sharon, Conn.

September 22—"Safeguards for Raw Milk," Dr. Albert L. Brown, Deposit.

October 6—"Facing Animal Disease on the Farm," Dr. John F. Miller, Albany.

October 20—"Anthrax," Dr. Charles Linch, Assistant Director, Bureau of Animal Industry, New York State Department of Agriculture and Markets, Albany.

November 3—"Shipping Fever, or Septicemia Hemorrhagica," Dr. George A. Knapp, Millbrook.

November 17—"Common Diseases of the Feet of the Horse," Dr. Wm. Henry Kelly, Albany.

Programs scheduled for December and January are as follows:

December 1—"Classifying the Dairy Herd," Dr. H. E. Hodges, Borden's Farm Products Co., New York City.

December 15—"Barnyard Dentistry (Care of the Teeth of the Horse)," Dr. A. L. Smith, Mechanicville.

December 29—"Live Stock Cooties (Lice on Farm Animals)," Dr. B. J. Cady, U. S. Bureau of Animal Industry, Albany.

January 12—"The Stallion Law," Dr. J. G. Wills, New York State Department of Agriculture and Markets, Albany.

January 29—"The Cow Takes an Examination (The Importance of Physical Examination of the Dairy Cow)," Dr. J. J. Regan, Dairymen's League, New York City.

These programs, of general as well as professional interest, are broadcast every two weeks, on Friday, at 12:30 p. m., E. S. T.

Regular Army

Major Everett C. Conant is detailed as a student under the provisions of section 127a, National Defense Act, as amended, in addition to his other duties, for the purpose of pursuing a course of instruction in the investigation of "Animal Diseases," at the University of Kentucky, Lexington, Ky.

Each of the following-named officers of the Veterinary Corps is relieved from further assignment and duty at the station as specified after his name, effective at such time as will enable him to comply with this order, will then proceed to Carlisle Barracks, Pa., and report to the Commandant, Medical Field Service School, on or about December 29, 1933, for duty for the purpose of pursuing the basic course of instruction:

2nd Lt. Wayne O. Kester, Fort Hayes, Ohio.
2nd Lt. Clarence L. Taylor, Fort Snelling, Minn.

Veterinary Reserve Corps

New Acceptances

Carter, Thomas Byron..Capt.....Gresham, Ore.
Whitlock, Raymond Francis..Capt.....Monett, Mo.

Kentucky Tuberculosis-Free

Kentucky has eradicted bovine tuberculosis. The Blue Grass State was added to the honor roll on November 1, as the twelfth state to win this distinction. It took fifteen years to clean up the Kentucky herds, even though the extent of the disease was not great, except in the vicinity of some of the large cities. The honor roll now is as follows:

North Carolina	Idaho
Maine	NorthDakota
Michigan	Nevada
Indiana	New Hampshire
Wisconsin	Utah
Ohio	Kentucky

12th International Veterinary Congress
New York—August 13-18, 1934

ORGANIZATION OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION, 1933-1934

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Waldorf-Astoria Hotel, New York, N. Y.
August 13-18, 1934

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She Needed Minerals

What does a cow eat in her spare moments?

Perhaps all cows are not so catholic in their tastes as the one killed recently in a Rotterdam slaughter-house, but here is a list of things found in the stomach of the Dutch cow: 27 medium-size nails, two four-inch nails, two gas-meter token coins, ten coins of Dutch mintage, four pieces of copper and four drawing-pins. At the time she was killed, the cow was in excellent condition, says the *Veterinary Record.*

FREE WHEELING IN THE TUBERCULOSIS MOVEMENT

By H. E. KLEINSCHMIDT

Steadily, for more than a quarter of a century, the number of deaths from tuberculosis has decreased. In 1900, the death-rate from this disease was 202 per 100,000 population. This rate has declined on an average of about 4½ points each year, so that, in 1930, the rate was 71 per 100,000 population. Until the beginning of the depression, this favorable trend was attributed by health officers mostly to a steadily rising plane of living of the American people. Other factors, of course, were given due credit, but what could be more logical than to assume that tuberculosis, a disease known for centuries to go hand in hand with poverty, should decline with the rise in wages, better housing, more abundant food and more leisure time?

But suddenly our prosperity buckled a wing or something, and spun off into the most desperate depression this country has ever seen. No wonder that from various quarters one heard the prediction that the tuberculosis death-rate would rise. Actually a strange thing has happened. During the four years of depression, deaths from this disease have continued to decline. There stands the record of the past four years. It gives us no inkling of what may happen in the future but it is a strange phenomenon. How can we account for it? Have our previous teachings been wrong or is there something unusual about this particular depression?

Of all the explanations offered, the one which seems most probable is that the momentum of the tuberculosis movement has been carrying us through just as the free-wheeling device on the auto enables the car to speed on for some distance with a minimum consumption of gas. Thirty years ago, tuberculosis

was permitted to run its course almost unmolested. Then, in 1904, a few courageous doctors dared to challenge the old enemy. That set in motion the popular movement which since then has built in this country more than 600 sanatoria, established diagnostic clinics in nearly every community, mobilized an army of public-health nurses and broadcast knowledge about tuberculosis to the remotest corners of the land. Thanks largely to the money derived from the sale of Christmas seals, the movement gained power year by year. Once going, even the drastic economics of these recent times apparently could not stop the good effects.

How long the tuberculosis movement can continue on free wheeling no one knows. Certainly it cannot be indefinitely. In an emergency the American people can do great things. Health departments and sanatoria have continued to function with meager funds. Cheap food, low wages and the loyalty of employes have enabled them to do so. Some doctors and nurses have worked on for nothing more than board and lodging. But that cannot go on. Sooner or later sanatoria, if not properly supported, will have to close their doors. The patients will be thrown back on their helpless communities soon to die. Well people compelled to live with them will be in danger of getting the disease. Without the watchful care of the public health nurse the control of the spreaders will relax and more disease will follow. And so with the other measures necessary to keep a controlling hand over tuberculosis. On an up-grade, free wheeling is no good whatever.

Health is won by long-time planning. Tuberculosis does not kill immediately. Nor can it be kept down by occasional spurts of energy. Only by steady progress can we hope to overcome it.

The object of tuberculosis associations is to keep the tuberculosis machine running—powerfully and smoothly. Lost momentum must be regained. The end of the hard journey to the place where every family can be assured of safety against tuberculosis is not yet in sight. If we roll back now our children will have to re-travel much of the hard-earned progress won in the past, and in the meantime many of them will sacrifice their lives. Harsh economy must no doubt still be practiced but, when health is at stake, penny-pinching is expensive business and leads only to more expense. A quick pick-up is the order of the present day. Christmas seals stimulate health work of all kinds and furnish the power to carry on the fight against tuberculosis.

Hospital Owners Organize

The American Animal Hospital Association was organized at Chicago, November 9-10, 1933, and the following officers were elected to serve the organization: President, Dr. M. L. Morris, Stelton, N. J.; vice-president, Dr. D. A. Eastman, Moline, Ill.; secretary-treasurer, Dr. A. R. Theobald, Cincinnati, Ohio. The first regular meeting will be held in Cincinnati, Ohio, some time during April, 1934.

The objects of the new association are:

1. To provide the best possible veterinary service and hospital facilities for the care and treatment of dogs, cats and other pets, including caged birds.

2. To protect and promote the professional interests of veterinarians engaged in the hospitalization of small animals.

3. To establish and maintain a high standard for the hospital, the equipment, the personnel and the methods employed.

4. To disseminate helpful information among owners of small animals, to neutralize the ill effects of widely circulated misinformation, pertaining to the health of small animals and the treatment of their ailments.

5. To coöperate with the Section on Small Animals of the American Veterinary Medical Association.

6. To coöperate with our colleges in elevating the standards of veterinary education.

7. To promote progress and coöperation between small-animal hospitals.

Fifth World's Poultry Congress

Dr. Evan L. Stubbs, Professor of Veterinary Pathology, represented the University of Pennsylvania and the Pennsylvania State Veterinary Medical Association at the Fifth World's Poultry Congress, held September 6 to 15, 1933, at Rome, Italy. This Congress has for its purpose the bringing together, from all parts of the world, of people who are interested in poultry. The meeting was held under the patronage of His Majesty, King Victor Emmanuel III, and His Excellency, Premier Benito Mussolini. Premier Mussolini opened the Congress, which consisted of scientific meetings and the International Poultry Exhibition. At the scientific meetings 25 general reports and 147 section papers were presented. Dr. Stubbs presented the subject of "Fowl Leukosis," based on work done by him at the University of Pennsylvania.

Papers were delivered in five official languages: English, German, French, Italian and Spanish.

The Congress was very well attended, the exhibition being held in the old Trajan Markets, which were put into condition for this event at the order of Premier Mussolini. It is said that chickens and eggs were traded in these markets at least two centuries before the Christian Era. The scientific meetings were held in the International Institute of Agriculture, in Rome.

Several trips were made in connection with the Congress, including visits to provincial poultry farms, the excavations at Ostia, Mussolini's reclamation projects at Littoria and Maccarese, the Pontifical Villa, and Tivoli. At the close of the session there was a tour especially arranged for members of the Congress. The tour included visits to Naples and Pompeii, Perugia, the Santa Lucia poultry and rabbit farm, and the famous chocolate factory at Perugina. Florence, Pisa, Leghorn and the Leghorn country, Bologna, Villa Ghigi, Rovigo, Padua, Venice, Murano, Burano, Vincenza, Valdagno, Milani, and Como were points of interest on the tour.

Dr. Stubbs gave reports about his trip to the Keystone Veterinary Medical Association, on October 25, the Pennsylvania State Veterinary Medical Association, on October 26, and the Veterinary Medical Association of New York City, on November 8.

A. H. C.

County Agent Hopper Cooperates

Knox County, Missouri, has an extension agent who believes in coöperation. His name is O. J. Hopper, and his policy of cooperating with practicing veterinarians is set forth in the following letter, addressed to the farmers in Knox County the past fall.

> Unless your hogs have been vaccinated for hog cholera with the double treatment they are in danger. Hogs have been lost on many farms already.
>
> Observe the following precautions:
>
> Consult your local veterinarian before your hogs get sick.
>
> Don't bring new hogs to your farm.
>
> If necessary to bring strange hogs to your farm, quarantine them away from your other hogs for at least two weeks.
>
> Don't purchase hogs which are offered for sale by any speculator, dealer, or any other person engaged in the business of assembling, reselling or trafficking in swine unless the proof can be given that said hogs have been vaccinated with the double treatment prior to three weeks before the sale.
>
> Your local veterinarian is an authority on hog cholera and he will be glad to help in stamping out this plague.

Mr. Hopper believes in the policy, "Live and let live."

CENTRAL NEBRASKA VETERINARY ASSOCIATION

A meeting of the Central Nebraska Veterinary Association was held at Kearney, October 27, 1933, with about 75 veterinarians in attendance.

The chief topic for discussion was "Equine Encephalomyelitis." Drs. Karl F. Meyer and C. M. Haring, of the University of California, were among the distinguished veterinarians present and both spoke on various phases of encephalomyelitis. Among the speakers who contributed to the discussion of this topic were Drs. C. E. Mueller, of Ogallala, Max Lentner, of Superior, Dr. A. E. Hasselbalch, of Saint Edwards, and Dr. L. F. Nisley, of Gothenburg.

Other prominent visitors who attended this meeting were: Dr. C. P. Fitch, president of the A. V. M. A.; Dr. I. E. Newsom, of the Colorado Agricultural College; Dr. H. L. Feistner, state veterinarian of Nebraska, and Dr. A. H. Francis, U. S. Bureau of Animal Industry inspector-in-charge at Lincoln, Neb.

The election of officers for the ensuing year resulted as follows: President, Dr. J. S. Anderson, Hastings; vice-president, Dr. Paul Baker, Elm Creek; secretary-treasurer, Dr. C. E. Mueller, Ogallala.

CENTRAL NEW YORK VETERINARY MEDICAL ASSOCIATION

The twenty-fourth semi-annual meeting of the Central New York Veterinary Medical Association was held at the Onondaga Hotel, Syracuse, November 2, 1933. The meeting was called to order by the President, Dr. M. W. Sullivan. Roll-call showed 24 members in attendance. Three new members were received into the Association at this time, and there was one reinstatement.

Dr. M. F. Wilkinson, of Utica, gave an interesting paper on Bang's disease, which was discussed at length. This was followed by a talk by Dr. A. A. Brockett, of Little Falls, on his methods of treating fistula of the withers, poll-evil and quittor.

Dr. A. H. Ide, of Lowville, gave a short explanation of his use of compound calcium gluconate solution intramuscularly for the treatment of parturient apoplexy in cows. Dr. Ide uses a 50 per cent solution which he prepares in his laboratory. Dr. W. L. Clark, of Seneca Falls, presented a paper on the treatment of open joint and fistula of the withers by the use of tolamin.

Dr. A. K. Zellner, of Oneida, gave a short talk on his observations in mastitis, especially concerning the relative merits of the thybromol test and physical examination. While the former test is good, he said, too much reliance can not be placed on it alone. He urged the desirability of coöperating with boards of health in the physical examination of cattle, with special reference to mastitis. A discussion by Dr. J. N. Frost, of Ithaca, followed. He mentioned the method of handling three-teated cows by the removal of the teat of the affected quarter. Dr. J. C. Stevens, of Cortland, offered suggestions, and Dr. J. K. Bosshart, of Camden, gave an interesting discussion of the subject.

The ladies joined the gentlemen for the dinner. After a few preliminary remarks by Dr. H. J. Milks, Dr. Frost, as the speaker of the evening, was given the floor, and delivered a splendid address on "Fungi Poisoning."

W. B. SWITZER, *Secretary.*

NEW ENGLAND VETERINARY MEDICAL ASSOCIATION

The fourth annual meeting of the New England Veterinary Medical Association was held in Boston, Mass., November 6-7, with more than one hundred veterinarians contributing to the success of the gathering. Morning sessions were held at the Hotel Bradford and afternoon clinics at the hospital of Dr. William Simpson, in Malden, Mass.

The morning session on the first day was opened with an address by the President, Dr. Edwin T. Laitinen, of West Hartford, Conn. The first paper at this session was given by Dr. J. F. Witter, of the University of Maine, on "Internal Parasites in Horses." Discussion on this paper was opened by Dr. A. J. Neal, of Bangor, Me. "Unusual Experiences Treating Wild Animals" was the title of a paper by Dr. H. L. Mills, of Burlington, Vt. An illustrated paper on "Some Diseases of the Conjunctiva and Cornea," by Dr. Theodore L. Terry, of the Massachusetts Eye and Ear Infirmary, Boston, was enjoyed greatly.

The program of the section day included the following features:

An illustrated lecture on "Equine Encephalomyelitis," by Lt. Col. R. A. Kelser, V. C., U. S. A., Boston; an illustrated paper on "The Symptoms and Lesions Produced by Various Poisonous Plants," by Mr. A. B. Clawson, of the U. S. Department of Agriculture, and a paper on "Skin Diseases," by Dr. George Ludins, of Hartford, Conn. A round-table discussion was led by Dr. J. F. DeVine, of Goshen, N. Y.

Dr. N. J. Pyle, of Pearl River, N. Y., was present to outline the plans for the coming Twelfth International Veterinary Congress and to appeal to the veterinarians present to become members. Dr. O. E. McKim, of Port Chester, N. Y., also spoke in behalf of the Congress, urging all veterinarians to support the undertaking.

Election of officers for the year resulted as follows: President, Dr. G. N. Welch, Northfield, Vt.; vice-presidents, Drs. N. H. Tenney, White River Junction, Vt., A. J. Neal, Bangor, Me., F. V. Barrett, Peterboro, N. H., R. G. Lawton, Providence, R. I., C. A. Burnette, Winsted, Conn., and B. S. Killian, Somerville, Mass.; secretary-treasurer (reëlected), Dr. H. W. Jakeman, Boston, Mass.

H. W. JAKEMAN, *Secretary.*

CHICAGO VETERINARY MEDICAL SOCIETY

At a meeting of the Chicago Veterinary Medical Society, Chicago, Ill., on October 10, 1933, Dr. Arthur D. Bissell, of the Surgery Department, Billings Memorial Hospital, University of Chicago, presented an interesting paper on the subject: "A Comparative Study of the Anatomy of Hernias in Animals and in Man."

MICHIGAN-OHIO VETERINARY MEDICAL ASSOCIATION

The semi-annual meeting of the Michigan-Ohio Veterinary Medical Association was held at Adrian, Mich., November 9, 1933, with an attendance of more than 75, including the junior and senior veterinary classes from Michigan State College. The President, Dr. S. G. Colby of Monroe, Mich., presided.

The program was opened by Dr. T. A. Sigler, of Greencastle, Ind. His first remarks pertained to the part played by the veterinary profession in the control of diseases. He then went on to discuss such subjects as periodic ophthalmia, laminitis, azoturia, navel-ill and exostoses, and outlined new methods for treating these conditions. It is evident, he said, that the veteri-

narian must keep pace with new developments in the field of veterinary science; otherwise, he will be eclipsed by the new generation of veterinarians.

In the afternoon, Dr. Sigler conducted a clinic in a barn, which depicted the usual conditions under which a country practitioner has to work. Dr. Sigler operated on two valuable horses, both of which were roarers. The ease and simplicity with which he did his work inspired every veterinarian present. His demonstration of a docking operation was worthy of emulation. He also demonstrated epidural anesthesia, after which he did the tail-setting operation. His method of facial nerve blocking and nerve blocking of the foreleg appeared simple after his demonstration.

The attendance of practitioners was about equally divided between Michigan and Ohio veterinarians.

E. C. W. SCHUBEL, *Secretary.*

CEDAR VALLEY (IOWA) VETERINARY ASSOCIATION

The Cedar Valley Veterinary Association met at Waterloo, Iowa, November 13, 1933, with 26 practitioners from Floyd, Chickasaw, Bremer, Black Hawk, Buchanan, Linn, Tama, Hardin and Benton counties, Iowa, in attendance.

The feature of the program was a discussion of the relationship between hog influenza and human influenza by Dr. R. E. Shope, of the Rockefeller Institute of Medical Research, Princeton, N. J. Others who participated in the program were: Drs. R. M. Hofferd, federal inspector in charge of hog-cholera control, Cedar Rapids; A. R. Menary, milk inspector for Cedar Rapids; P. V. Neuzil, of Blairstown; L. F. Bacon, of Cedar Falls; F. C. Roach, of Nashua; W. L. Hanson, of Aplington; P. C. Molgard, of Eldora; C. C. Graham, of Wellsburg; G. Lames, of Dysart; H. S. Lames, of La Porte City; H. J. Nygren, of Waverly, and Dick N. Voetberg, of Traer.

WEST VIRGINIA VETERINARY MEDICAL ASSOCIATION

The annual meeting of the West Virginia Veterinary Medical Association was held in Charleston, October 18-19, 1933, with about 50 veterinarians in attendance. Out-of state speakers were Dr. L. A. Merillat, of Chicago, Ill., and Dr. B. Scott Fritz, of Harrisburg, Pa. The West Virginia Commissioner of Agriculture, Mr. J. B. McLaughlin, also spoke to the group.

J. H. RIETZ, *Secretary.*

PURDUE UNIVERSITY SHORT COURSE

The 1933 Veterinary Short Course was held at Purdue University, November 14, 15 and 16. About 100 veterinarians were present.

The afternoon session, November 14, was devoted to a discussion of postmortem findings in poultry, hogs, sheep, cattle and horses brought to the veterinary laboratory. The following diseases were discussed. Poultry: paralysis, pullorum disease, tuberculosis and tumors. Hogs: cholera, influenza, anemia and pox. Horses, cattle and sheep: swamp fever, influenza and internal parasites. This was followed by a discussion by Dr. A. L. Delez on the results of his experimental work in attempting to produce immunity against Bang's disease, by injecting calves with bovine strains of *Brucella abortus*. The results show that a definite immunity has not been produced.

The following diseases were demonstrated at the poultry clinic: Paralysis, tumors, bronchitis, fowl typhoid, tuberculosis and pullorum disease. Diagnostic tests were demonstrated as follows: Serological tests for pullorum disease (both the slow and the rapid) and the intradermal tuberculin test for avian tuberculosis. A number of veterinarians were qualified for pullorum testing.

The program in the afternoon, November 15, was as follows: Mr. Leroy L. Jones discussed problems in the control of pullorum disease from the hatcheryman's point of view. He emphasized the importance of good testing, the advisability of making two tests of breeding flocks during the hatching season, and the danger of introducing the disease into tested flocks through the purchase of breeding flocks.

Dr. H. Preston Hoskins, secretary of the American Veterinary Medical Association, discussed the importance of a larger membership in the A. V. M. A., and the advantages of becoming a member at this time, because of the joint meeting of the Twelfth International Veterinary Congress and the A. V. M. A. in 1934. He also touched on veterinary education, pointing out that the number of students who would be graduated from our veterinary colleges in the next four years would not quite equal the estimated needs for veterinarians.

The present economic situation, factors responsible for variations in prices of agricultural products, the revaluation of the dollar, and return to the price level of the '20's was discussed by Prof. Lynn Robertson. Major C. W. Greenlee, V. C., U. S. A., Fort Benjamin Harrison, and Dr. D. M. Campbell, of Chicago, discussed osteomalacia, giving the cause and symptoms, with the

history of outbreaks in army horses, both in the United States and in the Philippines.

Not all of the surgical cases could be operated on in the time allowed for the clinic. Two operations may be mentioned—the enucleation of the eye of a horse, and the return of a prolapsed rectum in a beef cow. Dr. T. H. Ferguson, of Lake Geneva, Wis., operated on these cases. Dr. W. B. Craig, of Indianapolis, autopsied several lambs that had died in the feed-lot from dietetic causes. He took this opportunity to demonstrate correct methods of performing an autopsy, and discussed the different pathological conditions that may occur in sheep.

Prof. O. C. Lee discussed plants poisonous to live stock, and by the use of lantern-slides showed how to recognize plants commonly responsible for live stock losses in Indiana. Mr. G. P. Snow, of Chicago, discussed the recent outbreaks of encephalomyelitis in horses which occurred in California and Nevada. Moving pictures of horses that had the disease, and methods of producing a protective serum were shown.

The dinner meeting was held in the Memorial Building. Dr. H. Meade Hamilton, of Muncie, the toastmaster, called on a number of veterinarians for short addresses. Dr. J. L. Axby, State Veterinarian, was the principal speaker. He discussed the responsibilities of the State Veterinarian and the Live Stock Sanitary Board, the splendid service that veterinarians had rendered, and the coöperation that had been given him since he became State Veterinarian.

R. A. CRAIG.

NEVADA STATE VETERINARY ASSOCIATION

The mid-year meeting of the Nevada State Veterinary Association was held at the Department of Veterinary Science, of the University of Nevada, Reno, September 27, 1933. The meeting consisted of a brief session for the transaction of necessary routine business. No regular program was presented, but equine encephalomyelitis was discussed informally, with Dr. L. R. Vawter, of the University of Nevada, leading the discussion.

WARREN B. EARL, *Secretary.*

STATE BOARD EXAMINATION

Kansas State Board of Veterinary Medical Examiners. State House, Topeka, Kan. January 9, 1934. Dr. Thos. P. Crispell, Secretary, Parsons, Kan.

NECROLOGY

JOHN WILSON HILLMAN

Dr. John W. Hillman, of North Adams, Mass., died September 23, 1933, following an operation for acute appendicitis. He was stricken on September 16 and was operated upon immediately, but peritonitis and gangrene already had set in, and he died a week later, after a period of intense suffering.

Born November 14, 1908, at Heath, Mass., he attended and was graduated from the Arms Academy before entering the School of Veterinary Medicine at the University of Pennsylvania. Following his graduation in 1932, he located at North Adams, and was in general practice there at the time he was stricken.

Dr. Hillman joined the A. V. M. A. early in the current year. He was a member of Alpha Chapter of Omega Tau Sigma Fraternity. Surviving him are his mother, father and two sisters. He was buried from the home of his parents at Shellburne Falls, Mass.

To those who have known this young practitioner, this report comes as a painful shock, and all are united in expressing their sympathy toward the bereaved family.

A. H. C.

WILLIAM J. HART

Dr. William J. Hart, of Wetmore, Kan., died at his home, October 7, 1933. He was a graduate of Kansas City Veterinary College, and had been a member of the A. V. M. A. since his graduation in 1907. In that same year he also started to practice his profession in Wetmore, Kan., where he remained until his death. Dr. Hart operated a farm in addition to his veterinary practice.

EDWARD PUGH

Dr. Edward Pugh, of Alvin, Texas, died July 24, 1933. He was a graduate of the Kansas City Veterinary College, class of 1906, and formerly practiced at Lawton, Oklahoma, and Missouri City, Texas.

FRED NELSON WINCHESTER

Dr. Fred N. Winchester, of Kinsley, Kan., died in a hospital at Hutchinson, Kan., June 22, 1933. He was born at Larned, Kan., January 28, 1888, was graduated from the Larned High School and entered Kansas State College. He spent two years at Manhattan and then transferred to the Saint Joseph Veterinary College, from which he was graduated in 1909. He practiced at Hutchinson and at Burdett, Kan., before the World War. Dr. Winchester held a commission as Captain in the Veterinary Corps during the War and, following his discharge, entered practice at Kinsley, Kan. He is survived by his widow (née Jessie Newhouse), two sons and his mother. He was an Elk, a Mason and a member of the American Legion.

ALBERT JAMES BROWN

Dr. Albert J. Brown, formerly of Fayetteville, Tenn., died at Petrolea, Ontario, Canada, October 17, 1933. He was a graduate of the Ontario Veterinary College, class of 1907, and practiced in Fayetteville, Tenn., for 25 years. Last April, when his health failed, he returned to his former home in Canada, where he had been ill since. He is survived by his widow (née Eva Gaskill), one daughter, one son and his mother.

PRESLEY M. SELF

Dr. Presley M. Self, of Farmersburg, Ind., died at his home, October 28, 1933, the result of paralysis. He had been in failing health for some time.

Born in Greensburg, Ind., February 27, 1863, Dr. Self was a graduate of the Indiana Veterinary College, class of 1909. He had practiced at Farmersburg until his final illness. He is survived by his widow (née Minnie F. Graham), two daughters, one son, one sister and two brothers.

Dr. Self joined the A. V. M. A. in 1912.

HAROLD N. GUILFOYLE

Dr. Harold N. Guilfoyle committed suicide at the Connecticut State Prison, at Wethersfield, November 6, 1933, by jumping 50 feet from the top tier of the main cell block. He landed on his head on a flagstone floor and was dead when guards reached him.

Dr. Guilfoyle had been convicted of murder in the second degree, following a shooting affair in Hartford, Conn., several years ago. In June, 1932, he petitioned that his conviction be changed to first-degree murder and that he be sentenced to death by hanging, as a "humanitarian and merciful act."

Born in Philadelphia, Pa., January 9, 1885, Dr. Guilfoyle attended local schools and the University of Pennsylvania. He transferred to the United States College of Veterinary Surgeons, in Washington, D. C., and was graduated in 1907. He entered the service of the U. S. Bureau of Animal Industry, July 1, 1907, and had various assignments on tuberculosis eradication, meat inspection, tick eradication, and foot-and-mouth disease eradication. His service with the B. A. I. was terminated by resignation, May 20, 1929.

Dr. Guilfoyle joined the A. V. M. A. in 1918.

WILFRED ERNEST ARMSTRONG

Mr. W. E. Armstrong, M. R. C. V. S., of Wigton, Cumberland, England, died, October 26, 1933.

Born at Maryport, Cumberland, England, October 4, 1892, Mr. Armstrong received his preliminary education in English schools and studied veterinary medicine at the Ontario Veterinary College. He received his B. V. Sc. from the Canadian institution in 1915, and subsequently took up studies in the School of Veterinary Science of the University of Liverpool, from which he received his diploma of membership (M. R. C. V. S.) in 1920. He held a diploma also in veterinary hygiene (D. V. H.) from the same institution.

Mr. Armstrong joined the A. V. M. A. in 1922.

STONEWALL JACKSON SHIRLEY

Dr. S. J. Shirley, of Douglas, Georgia, died at his home, September 28, 1933, following a major operation performed a week previously. He was a graduate of the University of Georgia, class of 1924.

Following his graduation Dr. Shirley located at Douglas, where he built up a lucrative practice. He served for a period as Assistant State Veterinarian in his locality, but his major interest in veterinary matters was always along the lines of general practice, in which he was more than ordinarily successful.

Dr. Shirley was a member of the Georgia State Board of Veterinary Medical Examiners and of the Georgia State Veterinary Medical Association.

A. G. G. R.

WILLIAM HODGSON RIDGE

Dr. W. H. Ridge died at his home in Somerton, Pa., November 15, 1933, as the result of a stroke which occurred in 1921. He had been confined to his bed for the past ten years.

Born at Trevose, Pa., in 1853, on a farm that has been in the Ridge family for the past 140 years, Dr. Ridge studied veterinary medicine at the University of Pennsylvania. He was graduated in 1888 as a member of the second class in veterinary medicine to be graduated from that institution. He spent the balance of his active life in managing his farm in conjunction with an extensive veterinary practice. He continued his studies in agriculture and veterinary medicine and had exceptional ability in both fields. From 1890 to 1894, Dr. Ridge was an instructor in obstetrics in the School of Veterinary Medicine, at the University of Pennsylvania. Later he was selected by the Pennsylvania State Live Stock Sanitary Board to demonstrate the field diagnosis and treatment of sterility in dairy cattle. In 1895, the Veterinary Practice Act of Pennsylvania was amended to provide for a State Board of Veterinary Medical Examiners. Dr. Ridge was one of the five members constituting the first board. He was reappointed to the Board by numerous governors of Pennsylvania and served as President of the Board for many years.

Dr. Ridge joined the A. V. M. A. in 1895, and, at the time of his death, was one of four members joining that year who are still on the roll. He was a member and past president of the Pennsylvania State Veterinary Medical Association and a member of the Keystone Veterinary Medical Association. He held membership in several agricultural organizations and took an active and prominent part in all professional and agricultural activities.

A lifelong member of the Society of Friends, Dr. Ridge always conducted himself in strict accordance with the high ideals of that faith. He was a member of the Masonic Lodge and, during his long confinement, frequently mentioned the many deed of kindness shown by the Lodge and its members.

Dr. Ridge is survived by his widow (née Ella Tomlinson), four sons and one daughter.

C. J. M.

VERNON D. SHUTTE

Dr. Vernon D. Shutte, of Hilo, Hawaii, died July 17, 1933. He was a graduate of the San Francisco Veterinary College, class of 1907, and joined the A. V. M. A. in 1913.

RICHARD KINGMA

Dr. Richard Kingma, of Chicago, Ill., died at his home, October 22, 1933, as the result of a heart affection from which he had suffered during the past two years.

Born in The Netherlands, December 25, 1874, Dr. Kingma had been a resident of Chicago for 54 years. He was graduated from McKillip Veterinary College in 1910, and developed a practice of considerable importance among frugal Dutch settlers on the far south side of Chicago, who were engaged in truck and dairy farming. When the city absorbed that rural district, Dr. Kingma turned his attention to the treatment of small animals.

Surviving Dr. Kingma are his widow, five daughters and five sons, one of whom was preparing to enter a veterinary college next year and follow in the footsteps of his father.

L. A. M.

PROF. JOSE LIGNIERES

Prof. José Lignières, of Buenos Aires, Argentina, died on October 20, 1933. He was a native of France and secured his veterinary education at the National Veterinary School, Alfort, France, at about the time the scientific achievements of Pasteur were becoming recognized. Lignières was a student of Nocard, who was one of the chief collaborators of Pasteur.

From 1890 until 1893, Prof. Lignières was an assistant at Alfort. Later he was appointed a department head and, following that, he was promoted to the position of Director of the Veterinary Service of Paris. In 1898, agricultural interests in Argentina requested Pasteur to send a scientifically trained investigator to that country to study certain diseases which were menacing the cattle industry there. Pasteur and Nocard selected Lignières for the assignment. The results of Lignières' investigations of various animal diseases in South America, including Texas fever, foot-and-mouth disease, hemorrhagic septicemia, mal de caderas, BCG vaccination, are well known to veterinarians.

Prof. Lignières then joined the Faculty of Agronomy and Veterinary Medicine of the National University of Buenos Aires. He

became Counselor in 1904, a position he retained until 1911. In 1909, he was appointed Professor of Bacteriology in the institution. He also founded and directed the Bacteriological Institute of the Ministry of Agriculture of the Argentine Republic.

Prof. Lignières was the Argentine delegate to many international scientific gatherings, including the Eleventh International Veterinary Congress, held in London, in 1930. His scientific achievements have received the attention of many foreign governments and scientific institutions. In 1926, he was designated a Knight of the Legion of Honor of France. He had many decorations conferred upon him by scientific organizations. He was the Argentine representative on the Permanent Committee of the International Veterinary Congresses, member of the Academy of Veterinary Medicine of Paris, corresponding member of the Medical Academy of Paris, and non-resident member of the Academy of Agriculture of France.

ARTHUR M. CLIFFORD

Dr. Arthur M. Clifford, of Temple, Texas, was murdered by an unknown person on or about November 8, 1933, the last day he was seen alive. The body was found in a patch of weeds, about two miles from town, November 14. He had been struck on the back of the head with a heavy, sharp instrument. The murderer was later apprehended and has confessed. Jealousy was the motive.

Dr. Clifford was a graduate of the Ontario Veterinary College, class of 1908, and was 58 years old. He had practiced at Temple since about 1910.

F. D. P.

FLOYD H. HICKS

Dr. Floyd H. Hicks, of Allegan, Mich., died November 15, 1933, at his hunting camp near Turin, Mich. Dr. Hicks was stricken on his arrival at the camp and death was attributed to a stroke of apoplexy. He was in his 56th year, and was a graduate of the Allegan High School and of the McKillip Veterinary College, class of 1911.

Dr. Hicks joined the A. V. M. A. in 1916. He had practiced in Allegan since the time of his graduation. He is survived by his widow and one brother.

JOSIAH STOKES

Dr. Josiah Stokes, of Elmhurst, Ill., died at his home November 14, 1933, after an illness of three months.

Born in England, September 17, 1861, Dr. Stokes was a graduate of the McKillip Veterinary College, class of 1913. He practiced at Byron, Ill., for some years but had been located at Elmhurst since about 1920.

Dr. Stokes joined the A. V. M. A. in 1922. He was a member of Elmhurst Lodge, A. F. & A. M. He is survived by his widow, three daughters and two sons, Drs. Albert J. Stokes (McK. '12) and Sidney W. Stokes (McK. '17), both of Chicago. A third son, Major W. J. Stokes (McK. '07), died in the Philippines, November 3, 1924.

CHARLES EDWARD SAYRE

Dr. Charles E. Sayre, of Norfolk, Neb., died of pneumonia, November 18, 1933.

Born at Madison, N. J., March 8, 1862, Dr. Sayre studied veterinary medicine at the Chicago Veterinary College. He was graduated in 1887 and practiced in Chicago for a number of years. He taught veterinary dentistry at the Chicago Veterinary College in the '90's. While practicing as a veterinarian, Dr. Sayre studied dentistry at Northwestern University. He also taught comparative anatomy in the dental school at Northwestern while he was a dental student. Later, he studied medicine at the Hering Medical College in Chicago and received his M.D. in 1894. He took another course in medicine at the College of Physicians and Surgeons of Chicago School of Medicine of the University of Illinois and received his degree from that institution in 1897. The following year, he took work at the Chicago Postgraduate School of Medicine. He was a professor in the Hering Medical College from 1905 until 1915.

Dr. Sayre joined the A. V. M. A. in 1890. He left Chicago to locate in Norfolk, Neb., some years ago and at the time of his death was a member of the Nebraska State Medical Association.

P. L. SIMMONS

Dr. P. L. Simmons, of Racine, Wis., a registered non-graduate practitioner, died November 20, 1933, following injuries received when an automobile he was driving crashed into a North Shore train at a crossing a short distance from Racine. He was 74 years of age.

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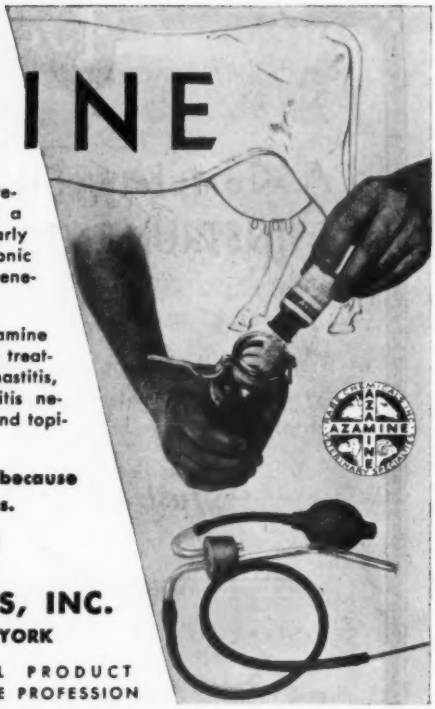

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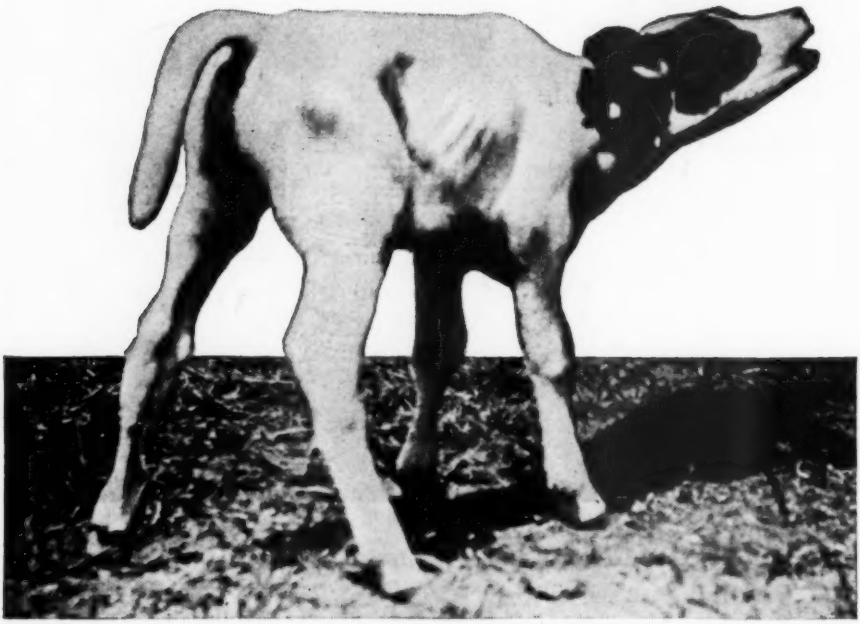

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(*b*) *the stamping out of foot-and-month disease, because it was deemed wasteful to destroy healthy animals to accomplish the purpose;*

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Meanwhile the Army, from Washington to Wilson, in refusing to utilize contemporary veterinary medicine, fulminated appalling epizoötics among animals in all of the wars of American history.

However, the veterinary profession has marched on and, as 1934 approaches, it looks back without rancor at the strange obstacles it had to roll from the path to its altruistic objective.

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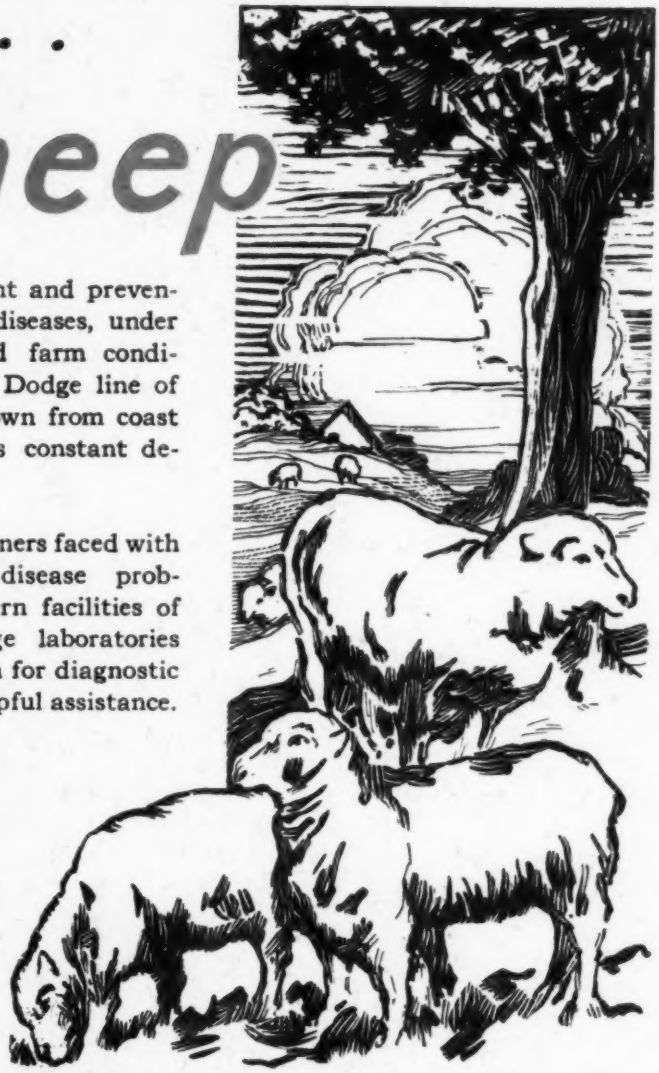

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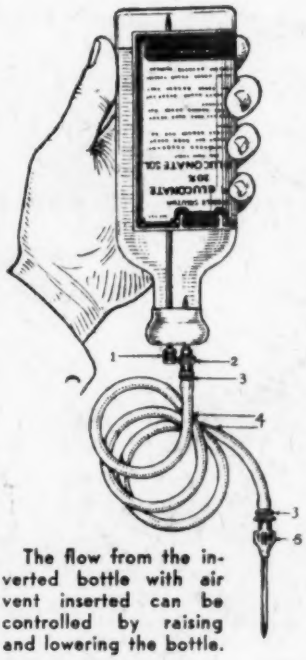

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